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Maryland

STATE MEDICAL JOURNAL

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UARY, 1961

ANNUAL MEETING—WEDNESDAY, THURSDAY, FRIDAY, APRIL 26, 27, 28, 1961

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MARYLAND
STATE MEDICAL JOURNAL
Volume 10 January 1961 Number 1

EDITORIAL

Prescriptions: Generic versus Trade Names

WE HAVE HEARD a great deal lately from various groups and individuals who are again making accusations of profiteering and "monopoly" in the prescribing of drugs. The medical profession has had its share of abuse in this matter because of statements to the effect that doctors should prescribe by generic terms rather than by trade names. These people argue that such type of prescription would provide the same result but at a lower cost to the patient. In the meantime, of course, if the physician prescribes by generic name, the manufacturer of the trade name drug, who developed his product to treat a certain illness and who expended considerable money to do so, must permit *all* drug manufacturers to reap the benefits of his developmental efforts.

The medical profession has not taken a specific stand on this matter, physicians having continued to write prescriptions according to their own preferences. Now we have received information which gives us considerable food for thought.

In a recent study conducted by a large ethical drug manufacturer, it was ascertained that ". . . the active ingredient content of the so-called 'generic equivalents' was found to fall outside the extreme of tolerance established by the USP in 35 per cent of the cases; whereas all samples of the branded products were of top quality." The study further revealed that "prescriptions written under the generic names netted the patient an average saving of 11 per cent, and another study for 15 leading ethical drugs for which generic equivalents are available showed savings to the consumer averaging only 12 per cent."

These figures refute the alleged enormous savings claimed by those who foster prescription by generic name and who would, in

effect, attempt to deprive the physician of his right to practice medicine to the best of his ability and according to his best judgment.

Armed with these facts, the physician can readily answer with good, solid reasons a patient who requests generic prescriptions, provided, of course, that the physician is opposed to granting such a request. Were such practice to become universal, it would place the pharmaceutical manufacturers in the position of sitting back and letting some one else develop new drugs. Thus, medicine's research programs for new therapeutic agents would be slowed to a snail's pace.

CALENDAR OF EVENTS

► Monday, January 16 ◄

PATHOLOGY SECTION, B.C.M.S.
8:00 P.M. University of Maryland
School of Medicine
"Cytology of Condyloma Accuminata,"
Z. Naib, M.D.
"Etiologic Factors in Spontaneous Cerebellar Hemorrhages," R. Levin, M.D., and J. Wagner, M.D.
"Kidney Lesions Following Administration of Versenate," M. D. Reuber, M.D.
"Diffuse Angiosis of the Liver in a 35 Year Old Female," R. Schultz, M.D.
Business Meeting.

► Tuesday, January 17 ◄

MARYLAND RADIOLOGICAL SOCIETY
Lord Baltimore Hotel
5:30 P.M. Film reading session.
(Please bring interesting cases.)
6:30 P.M. Cocktails, 7:00 P.M. Dinner,
8:00 P.M. Scientific Session.
"Carcinoma of the Lung," Eugene P. Pendergass, M.D., professor and head, Department of Radiology, Hospital of the University of Pennsylvania. All residents and other guests are welcome to attend the scientific and film reading sessions.

► Saturday, January 21 ◄

MEDICINE 1961
4:30-5:00 P.M. WMAR-TV
"Gall Bladder Disease,"
William F. Rienhoff, Jr., M.D.

► Tuesday, January 24 ◄

ANESTHESIA STUDY COMMITTEE
8:00 P.M., 1211 Cathedral Street

► Wednesday, January 25 ◄

HEART ASSOCIATION
Postgraduate Session
Hagerstown

► Thursday, January 26 ◄

OPHTHALMOLOGICAL SECTION, B.C.M.S.
6:00 P.M.

► Saturday, February 4 ◄

MEDICINE 1961
4:30-5:00 P.M. WMAR-TV
"Relationship Between Medicine and Industry,"
Ephraim T. Lisansky, M.D.

► Wednesday, February 8 ◄

MARYLAND SOCIETY FOR MENTALLY RETARDED CHILDREN
GREATER BALTIMORE CHAPTER
8:15 P.M., 2525 Kirk Avenue

► Saturday, February 11 ◄

MARYLAND TRUDEAU SOCIETY
Tidewater Inn, Easton, Md.
Scientific session on pulmonary disease

► Monday, February 13 ◄

SACRED HEART HOSPITAL
MEDICAL STAFF
11:30 A.M.
School of Nursing,
Bellevue Street, Cumberland

► Tuesday, February 14 ◄

MARYLAND SOCIETY ON ALCOHOLISM
Officers and Executive Committee
8:00 P.M. Council of Social Agencies,
22 Light Street

► Saturday, February 18 ◄

MEDICINE 1961
4:30-5:00 P.M. WMAR-TV
"Problems of the Newborn Baby,"
Alexander J. Schaffer, M.D.

YOUR MEDICAL FACULTY AT WORK

Executive Secretary
John Sargeant

The Executive Committee of the Council and the Council met on the dates indicated and took the actions shown:

Executive Committee, October 18, 1960

Designated Whitmer B. Firor, M.D., president, as official Faculty representative at the public hearing on the Faculty's proposed new fee schedule for workmen's compensation cases.

Executive Committee, October 27, 1960

1. Heard that the ophthalmologists are trying to "work out a plan for eye clinics in the various counties in the state."

2. Approved inexpensive pamphlets citing benefits of membership for enclosure with mailings to Faculty members.

3. Referred to the Committee to Study Problems of Mutual Interest to the Faculty and the Maryland Pharmaceutical Association a letter regarding proposals advanced by a pharmacist.

Council, November 15, 1960

1. Ratified legal defense for certain Faculty members.

2. Established meeting dates for 1961 Council meetings.

3. Heard a request to consider nominees for various positions on the Blue Cross and Blue Shield boards and committees, for action at the January meeting.

4. Established a policy that Faculty representation at national and/or regional meetings must be considered on an individual basis insofar as attendance of committee chairmen at such meetings is concerned, and payment of any expenses involved must be approved in advance by the Executive Committee.

5. Adopted a policy that meals not be paid for Faculty members, except staff members, attending committee or Council meetings.

6. Requested the Medical Economics Committee to reconsider the question of a Relative Value Schedule and to report back to the Council on this matter.

7. Authorized the treasurer to lend the Building Fund $30,000 to $40,000 from Faculty's cash reserves at a suitable rate of interest.

8. Authorized the Building Committee chairman to borrow up to $130,000 on a short-term loan from the Baltimore National Bank to complete the building renovations authorized by the 1960 House of Delegates at its annual meeting.

9. Requested the Building Committee chairman

to circulate a report to all members advising them of the building renovation progress and offering the suggestion to those who have not completed paying their assessment that payment in advance would reduce the sum necessary to borrow and thus reduce the interest obligations of the Faculty.

10. Authorized hiring of legislative personnel for the 1961 legislative assembly in Annapolis.

11. Approved April 4, 5, and 6, 1962 as the dates for that year's annual meeting.

12. Authorized purchase of the film, "I Am A Doctor," to assist in the medical career recruitment program.

13. Authorized the Committee on Public Instruction to discuss a medical career recruitment program with medical school and Woman's Auxiliary officials.

14. Authorized the Executive Committee to approve hiring of an actuarial consultant if the Fee Schedule Committee makes a specific request for such assistance.

15. Referred to the Baltimore City Medical Society a request for approval of an outpatient mental health project sponsored by Paul Lemkau, M.D.

16. Heard a summary of activity in connection with the work of Blue Cross/ Hospital Council and Faculty Conference Committee and made certain stipulations in connection with these discussions.

92 Cents of March of Dimes Dollar Spent In Maryland

The state of Maryland has been the principal beneficiary in the allocation of March of Dimes funds raised in the state over the past 23 years, it was disclosed recently in a financial summary prepared by The National Foundation.

More than 92 cents of every dollar from Maryland's March of Dimes has been used to aid the state's disease victims and in research projects conducted by Maryland institutions. Of the remaining eight per cent accruing to the national headquarters, a considerable amount returned to Maryland in the form of polio vaccine and gamma globulin and in other nationwide services conducted by The National Foundation.

The financial summary covers the period since the first March of Dimes was held in January 1938 and compares the net total of funds raised in the state with amounts made available to Maryland through September 30, 1960.

In this period, Maryland's 24 chapters of the March of Dimes organization raised a net total of $8,178,290.13 at an average fund raising cost of 10.5 per cent. Of this amount, $4,328,814.85 has been available to the county chapters in carrying out their extensive patient aid programs and includes advances of $837,040.19 from the national office to meet local emergency situations.

In addition, 54 grants totaling $3,177,342.86 have been made in support of research projects at Maryland institutions. Principal recipients of these grants have been: Johns Hopkins University, $2,816,478.69; the University of Maryland, $227,217.86 and Microbiological Associates $117,996.50.

Over and above the 92 per cent used by institutions and county chapters in the state, The National Foundation has financed within the state projects such as the historic field trials which proved the effectiveness of the Salk vaccine, epidemiological studies, and scholarship or fellowship grants to Maryland residents. National headquarters' expenditures for the vaccine trials in Maryland amounted to $43,954.46. In addition, the national office has sent into Maryland $79,177.77 worth of Salk vaccine and 61,668 cc's of gamma globulin in support of its polio prevention programs.

Two years ago, the National Foundation for Infantile Paralysis changed its name to The National Foundation and expanded its areas of interest to include birth defects and arthritis, using the scientific knowledge and experience gained in the fight against polio.

The 1961 March of Dimes takes place throughout the month of January.

REMINDER REGARDING RESOLUTIONS!

Important Notice for Component Medical Societies and Individual Members of Medical and Chirurgical Faculty

The House of Delegates of the Medical and Chirurgical Faculty approved the following recommendations concerning procedure governing reports and resolutions given at the Annual and Semiannual Meetings:

1. All reports must be sent to the Faculty office. Those reports which contain recommendations or resolutions must be in the office eight (8) weeks prior to the Annual or Semiannual Meeting, whichever happens to be concerned.

2. When the reports are received, those containing recommendations or resolutions will be sent to the Component Societies for consideration so that the Component Delegates may be instructed if desired. These reports will also be referred to Council for discussion at its meeting prior to Annual or Semiannual Meeting.

3. Those reports which contain resolutions are to be referred to the Resolutions Committee for consideration.

4. The Council will refer to the Resolutions Committee any recommendations which it feels should be formulated as resolutions. The Council will also transmit to the Resolutions Committee an opinion of the policy involved in the resolution.

5. Reports will be presented to the House of Delegates as usual, and it will be suggested as is normally done that reports not containing recommendations or resolutions be accepted as printed and distributed.

6. Those reports containing recommendations or resolutions will be considered and acted upon individually by the House of Delegates.

This policy will be followed in all future meetings.

AS A RESULT OF THIS ACTION OF THE HOUSE OF DELEGATES, RESOLUTIONS FOR PRESENTATION TO THE APRIL 1961 ANNUAL MEETING OF THE HOUSE OF DELEGATES, MUST BE IN THE HANDS OF THE SECRETARY, WILLIAM CARL EBELING, M.D., AT THE FACULTY OFFICE, BY MARCH 1, 1961.

As adopted by the Council, the members of the Medical and Chirurgical Faculty are advised that the Resolutions Committee is anxious to hear expressions of opinions from members on any resolutions being presented to the House of Delegates at either the Semiannual or Annual Meetings, and that members in good standing who might wish to appear before this Committee to discuss a pending resolution may do so upon making a request to that effect to the Resolutions Committee.

RESOLUTIONS FOR APRIL HOUSE OF DELEGATES MUST BE IN FACULTY OFFICE

BY

WEDNESDAY, MARCH 1, 1961

HEART ISSUE

The scientific papers in this issue have been provided by the Heart Association of Maryland. We are indebted to J. Frank Supplee, III, M.D. for assembling this material.

TEAR OUT THIS SHEET

To Order Patient Education Materials

One of the primary goals of the Heart Association's public education program is to help the physician in patient education. In this connection, the Association has a series of printed materials designed for physician-patient use. All of these materials are available in quantity from your local Heart Association. There is a 15¢ per copy charge for the Low Sodium Diet books, but all other materials are FREE.

Below is listed a sampling of materials written especially for physician-patient care.

☐ Please send me a sample of the 500 mg.

Your Sodium-Restricted Diet Books

Your 500 Milligram Sodium Diet 56 pages
Your 1000 Milligram Sodium Diet 56 pages
Your Mild Sodium Restricted Diet 48 pages

☐ Place my order for ..... copies at 15¢ ea.
Check enclosed.

Each booklet lists diets for three calorie levels. (A free sample of the 500 Milligram booklet is available from your Association. Additional copies are 15¢ each.)

Quantity ..... *Heart Disease and Pregnancy*—12 pages. For women with heart disease who are planning to have children.

Quantity ..... *Heart Disease Caused by Coronary Atherosclerosis*—20 pages. A doctor explains to his patient what happens during a heart attack and describes the healing process and treatment necessary for recovery.

Quantity ..... *High Blood Pressure*—16 pages. Explains what hypertension is and how the patient can cooperate with his physician.

Quantity ..... *Home Care of the Child with Rheumatic Fever*—26 pages. Provides practical pointers on home nursing techniques and suggestions for dealing with emotional problems.

Quantity ..... *If Your Child Has Rheumatic Fever*—16 pages. Discusses questions of care that arise during the acute and convalescent stages.

Quantity ..... *A Safe Work Load for Farmers with Heart Disease*—8 pages. Designed to help the farmer with heart disease and his doctor plan a work routine that is within the patient's physical capacity.

Quantity ..... *Strike Back At Stroke*—40 pages. Twenty-one therapeutic exercises to be prescribed by the physician are illustrated and described.

Quantity ..... *Strokes—A Guide for the Family*—20 pages. Designed to help families of patients understand strokes; offers practical suggestions for patient care.

Quantity ..... *Varicose Veins*—12 pages. Explains what varicose veins are and lists general recommendations for patients.

Please check *Publications and Visual Aids for Physicians*—A listing of Heart Association materials available for physician and physician-patient use.

NAME .....

ADDRESS .....

Tear out and send to: YOUR LOCAL HEART ASSOCIATION

or

THE HEART ASSOCIATION OF MARYLAND
415 NORTH CHARLES STREET
BALTIMORE 1, MARYLAND

An Appraisal of SURGERY For CORONARY HEART DISEASE

● *Experience with two operative procedures for coronary heart disease is reviewed, and a criteria for selection of patients for these procedures is presented.* ● *Preoperative and postoperative electrocardiograms, ballistocardiograms, and ballistocardiographic cigarette tests are reported in 34 patients who underwent phenol de-epicardialization and pericardiectomy. The operative mortality was 26 per cent, but these patients were severely ill and incapacitated and had not responded to medical therapy. Striking subjective improvement occurred in 14 of the 21 surviving patients in whom follow-up was performed. The objective studies were disappointing, not improving in a manner commensurate with the clinical improvement.* ● *Coronary endarterectomy was performed in six patients, of whom five survived and showed dramatic subjective improvement. It is too soon after operation (six months average) to have obtained adequate objective follow up.* ● *Patients in their fourth to sixth decade with severe angina not responding to vigorous medical therapy and not evidencing heart failure or massive cardiomegaly should be considered candidates for surgery. If coronary arteriography demonstrates complete occlusion of one major coronary artery with relatively satisfactory patency of the other and its major tributaries, endarterectomy should be considered; if the disease is severe and diffusely distributed throughout both coronary arteries and branches, de-epicardialization is feasible. In patients who are severely incapacitated and who do not meet these requirements for surgery, hypometabolic treatment would seem the logical choice.* ●

DESPITE MANY YEARS of surgical efforts and a multitude of technical approaches, surgery for ischemic heart disease remains a highly controversial subject. Conclusive evidence that *any* operative procedure increases effective coronary blood flow in the diseased human has never been presented. Unequivocal proof of increased survival in humans (with adequate simultaneous control patients) following operation is likewise lacking. The transfer of evidence from the experimental animal to humans in this realm is highly uncertain, because of the great pathological distinction between the widespread disease in humans and a normal coronary arterial tree in which a major artery is experimentally occluded (the remaining vessels not having the established network of collateral vessels found in the diseased human heart, and the large vessels lacking the atheromata of the human).

Frank W. Davis, Jr., M.D.

From the Department of Medicine, Johns Hopkins Hospital and Johns Hopkins University School of Medicine.

This study was supported in part by a Research Grant (H-327) from the National Heart Institute of the National Institutes of Health, United States Public Health Service.

Because the definitive evidence of efficacy (i.e., increased flow or increased survival) of surgery is not available, the clinician must use other criteria in making his decision to operate on patients with coronary disease. Several justifications for surgical therapy exist: (1) the belief that a procedure has theoretical justification, although not yet proven to be of benefit; (2) the belief that relief of pain might be accomplished, even though longevity might not be statistically prolonged; (3) pessimism regarding present medical therapy, especially in severely incapacitated persons. This reasoning omits all reference to the experimental animal, in which there is considerable evidence of increased survival after almost any prophylactic operation, which fact in itself leads to some doubt of the transferability of canine surgical studies to the clinic.

A review of all of the surgical approaches to coronary disease will not be undertaken here. A concise review of the anatomic pathologic problem has recently been published by Blumgart et al (1). These workers defined the problems of the various methods of increasing myocardial blood supply and suggested certain features which apply to the direct attack on the thromboatheromatous lesions. An earlier review by Hellerstein of the various surgical methods analyzed the problems of surgery considered, in general, the concepts underlying the surgical efforts (2). Hellerstein's conclusion was that no evidence exists of increased life expectancy in surgically treated patients, but he felt that justifictaion for surgical treatment on an experimental basis existed.

This report discusses two operative procedures. The results of surgery from the viewpoint of medical follow-up will be documented, and a discussion of our current philosophy on selection of patients for surgery will follow.

The first of the surgical techniques is a modification of phenol de-epicardialization as described by Harken et al (3). This technique resembles the older methods reported by Beck (4) and by Thompson (5), each having the general concept of creating an inflammatory reaction about the epicardial-pericardial surfaces, either to (a) increase collateral extra-coronary blood flow or (b) decrease the electrical excitability of the heart (and possible ventricular fibrillation) by diminishing the "current of oxygen differential" (6). The operation as performed in this series* has consisted of phenol de-epicardialization, sometimes with talc poudrage, in earlier cases with cardio-pneumopexy, and in later cases (where possible) with cardiomediastinopexy utilizing the mediastinal fat pad.

A smaller number of patients has been operated* on by using the direct approach of coronary endarterectomy, as performed also by Longmire (7) and Bailey (8). Of all surgical approaches to coronary heart disease, this seems the most physiologic in patients fulfilling the necessary criteria for operability.

PERICARDIECTOMY AND DE-EPICARDIALIZATION

THIS PROCEDURE has been performed on 34 adult patients of whom 27 were males and seven were females. The average age was 47.5 years. All had severe angina, 17 having angina with minimal effort and 17 also experiencing ischemic pain on frequent occasions at rest ("angina decubitus"). Thirteen had survived earlier myocardial infarction. In most of these patients, preoperative and postoperative studies were performed in the ballistocardiographic laboratory, where resting and post-smoking electrocardiograms and high-frequency ballistocardiograms were obtained by the techniques previously reported (9, 10). As in earlier studies, all records were classified as normal, borderline, or abnormal (or in cigarette tests as negative, borderline, or positive).

The ballistocardiographic patterns in the preoperative period were abnormal in 17 patients, borderline in six, and normal in six. Electrocardiograms were normal in 11, borderline in 4, and abnormal in 19. The smoking test was positive in 13, borderline in 5, and negative in 7.

Death occurred at the time of operation or within three months after in 9 of the patients. The surviving patients have been re-studied in the laboratory at periods ranging from three to 36 months after operation, an average of 18 months having elapsed in 18 of the 21 patients followed (three have been operated on in recent months and are not included). In the latest post-surgical laboratory survey, the BCG remained normal in two subjects, showed no change in two, improved in five, and became more abnormal in eight. The ECG remained normal in three, showed no change

*Operation in most instances was by David C. Sabiston, Jr., M.D.

in five, improved in three, and was more abnormal in seven. The cigarette test remained negative in two, showed no change in seven, improved in only one, and became more positive or more abnormal in two. (Table I)

Evaluation of clinical response is, admittedly, extremely hazardous; for most patients with angina pectoris improve with almost any therapeutic effort. On the other hand, these patients were all critically ill, having suffered repeated bouts of angina (10-30 or more daily in most cases), and *all* had been treated intensively by medical methods without real benefit. Improvement in a clinical sense, although largely judged by the patient's own estimate of exercise tolerance and need for nitroglycerin, represents a gain over extended therapy with program of bed rest, sedation, long-acting nitrites, and digitalis previously utilized in each patient.

Response in survivors is graded 1-4, with 1 representing slight or no improvement and 4 representing almost total freedom from angina with full mobilization and employability. Two late deaths (after six months) occurred. One patient, having shown transient symptomatic improvement, died suddenly at home six months after operation. The other made no initial improvement and was subjected to a second operative procedure, which revealed congenital atresia of the right artery and severe atherosclerosis of the left coronary artery.

Two patients have survived subsequent infarctions, both being symptomatically improved after operation and both being relatively more free of angina after the infarction than before surgery. Of the remaining patients, one showed 1+, five, 2+, ten, 3+, and four, 4+ improvement. (Table II) Thus, 41 per cent of the original patients on whom surgery was performed (60 per cent of the survivors) showed subjective improvement.

TABLE I

	Ballistocardiogram			Electrocardiogram			Cigarette Test		
	Normal	Borderline	Abnormal	Normal	Borderline	Abnormal	Negative	Borderline	Positive
Control	6(21 %)	6(21 %)	17(58%)	11(32%)	4(12%)	19(56%)	7(28 %)	5(20 %)	13(52%)
Following Operation	3(17.5%)	3(17.5%)	11(65%)	4(21%)	1(5%)	14(75%)	4(28.5%)	4(28.5%)	6(43%)

Tabular results of objective measurements in patients on whom de-epicardialization was performed. The control records were usually obtained immediately before surgery. The studies after operation as listed represent the longest follow-up in each patient from time of surgery. In several patients, records had improved and subsequently deteriorated, the latter being the results listed above.

CORONARY ENDARTERECTOMY

EXPERIENCE WITH this operation is limited, largely because of the strict criteria which we have established for selection of patients. We have performed it on six patients, all with severe angina, all males, with an average age of 44 years. Preoperative studies in the BCG laboratory show a lesser degree of abnormality, despite the evidence of almost complete (1) or complete (5)

TABLE II

Total patients operated	34
Operative death, or death within three months.	9(26%)
Patients lost to follow-up	2(6%)
Patients surviving three months or more	23(68%)
Late deaths	2(6%)
No symptomatic change	1(3%)
1–2+ Improvement	6(18%)
3+ Improvement	10(29%)
4+ Improvement	4(12%)

Summary of clinical results in patients operated by the technique of de-epicardialization. 1–2+ improvement indicates moderate relief of angina but with some restriction of activity and frequent nitroglycerin still necessary. 3+ improvement includes patients who have been rehabilitated, work full time, and take occasional nitroglycerin. 4+ improvement represents the change from complete invalidism to full time employment, with occasional or rare attacks of angina occurring. One patient in the 3+ group accomplished this degree of improvement only when surgery and I-131 thyroid ablation were combined.

obstruction to one of the two main coronary arteries with coronary arteriography. The preoperative BCG was abnormal in only two; the ECG, in two; and the cigarette test was positive in one of these six patients (at least one test was abnormal in each patient). Preoperative two step tests were performed in five patients and were positive (1.5 mm. ST segment depression) in four of them.

The patient in whom a left coronary endarterectomy was attempted died at operation; the other five, all with complete occulsion of the right main coronary artery, survived and clinically have show dramatic improvement. Unfortunately, the objective measurements have not yet been obtained in these subjects following operation. The average post-operative survival, at the time of this writing, is six months.

DISCUSSION

It is our unreserved opinion that the usual patient with mild to moderate angina pectoris is not a candidate for surgical therapy. Most patients with this disease respond well to the generally accepted conservative medical programs; therefore they need not be subjected to the risk and discomfort attending any operative procedure. Surgery has been utilized when all other efforts have failed. It is possible, largely on the basis of the experience reviewed above, to outline a tentative program for patient selection.

Direct endarterectomy seems to be the most reasonable technique, when considered on physiologic grounds. At present, it is felt that this operation is best performed in the rather unusual patient who (a) shows severe and unequivocal clinical angina; (b) is relatively youthful, probably in the fourth to sixth decade; (c) has evidence on adequate coronary arteriographic study of *complete* occlusion of one major coronary artery, with relatively less disease involving the opposite artery and branches; and (d) fails to respond to intensive medical therapy. The five successful endarterectomies reported here had no more morbidity associated with the operation than did the larger group of patients subjected to de-epicardialization, although one had a prolonged staphylococcal wound infection. Subjective improvement has been striking, and objective measurements in the near future will help to define further the success of this procedure.

In the patients studied before and after surgery by Longmire et al (11), the treadmill exercise tolerance increased, and it was felt that all had evidence of decreased myocardial ischemia. It seems reasonable to study all patients in this age group who have severe angina by arteriography and to consider direct surgical attack where complete occlusion of one artery is found.

The role of de-epicardialization is somewhat more difficult to define. Despite the high mortality rate in this series, it must be considered that all of these patients were desperately ill and that, although control patients were not studied simultaneously, these patients had been offered most

or all of the medical programs without improvement. The response to surgery in most survivors has led to the conclusion that this operation has a role in coronary therapy, in selected cases with intractable pain. The failure of objective studies to confirm the subjective improvement has been disappointing. In some measure, this might be explained by the creation of pericarditis, which adversely influences the resting ECG and BCG and probably is responsible for some or all of the graphic deterioration summarized here. It has been true that almost inevitably the ECG and BCG within two weeks after surgery have been profoundly abnormal, whereas with passing months, these abnormalities tend to decrease. Similar failure of the ECG to improve after operation has been reported by Brofman, although a small number of patients in his experience did show BCG improvement (12).

De-epicardialization is elected where (a) there is severe and incapacitating angina, not responding to medical therapy; (b) the patient is not beyond the seventh decade; (c) there has not been a recent infarct; (d) there is no congestive heart failure or massive cardiomegaly; and (e) the patient has such extensive atherosclerosis that endarterectomy does not seem safely possible. It is our hope that this operation might produce symptomatic relief and perhaps allow sufficient survival time for experimental metabolic efforts to induce whatever benefits are possible. In other patients, similarly incapacitated but not having an artery amenable to endarterectomy and not fulfilling the requirements for de-epicardialization, it would be reasonable to institute hypometabolic therapy with radioactive iodine.

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ETHICS CORNER

FROM TIME TO TIME this office receives requests for approval of feature articles about physicians who have performed some outstanding public service (generally outside their practice of medicine). These requests sometimes come from the newspapers, magazines, or other publishers of the article; sometimes from the physician himself.

While self-laudation of a physician is specifically prohibited under the Principles of Medical Ethics, it has been ruled that physicians who perform public service activities, such as with Boy Scouts, church work, or similar activities, may and should receive due credit for them. The Judicial Council of the AMA has stated, "The Judicial Council does not believe that the use of a physician's name in connection with a civic project should, in itself, be considered contrary to the Principles of Medical Ethics."

MANAGEMENT OF THE CHILDBEARING CARDIAC

Not one, but two lives are at stake when a woman with heart disease becomes pregnant. To sustain the cardiac patient through the ordeals of pregnancy, delivery, and the puerperium requires skillful and thoughtful management.

Henry J. L. Marriott, M.D.* and

Adrian S. Weyn, M.D.†

FIGURES DERIVED from more than a million pregnancies show the incidence of heart disease among pregnant women to be only about 1.38 per cent. On the other hand, heart disease has become a leading cause of maternal mortality during pregnancy and the puerperium, according to various authors, who attribute up to 27 per cent (1) of maternal deaths to heart disease. When encountered, therefore, it can be an ominous complication with a potentially fatal outcome for not one but two souls. Our purpose is to review some of the salient principles in the management of childbearing patients with heart disease. The care of such patients cannot be intelligently approached without a knowledge of the physiological changes that affect the cardiovascular system.

PHYSIOLOGICAL CHANGES

Toward the end of the first trimester, a number of cardiovascular adaptations begin to manifest themselves. Some of these progress to reach a peak level some eight to 14 weeks before term and then decline; others continue to progress through the second and third trimesters up to the time of delivery. The *heart rate* increases an average of 10 beats per minute by the 32nd week (2), which means an additional 14,400 contractions in the 24 hours. The *cardiac output* increases to a maximum of 40 per cent above normal by the 26th week (3), while the *plasma volume* increases by 22 per cent at the 32nd week (4). Heart rate, cardiac output, and plasma volume then decline until term.

Meanwhile, the *oxygen consumption* continues to rise to 15-18 per cent above normal and the minute *ventilatory volume* to 40 per cent above normal at term. Since the ventilatory volume exceeds the oxygen consumption, it is evident that the patient is hyperventilating; this is confirmed by a declining pCO_2. This respiratory inefficiency is most likely due to the increased work required to exchange air in lungs encroached upon by an expanding abdomen. The *vital capacity* remains relatively constant or, surprisingly, increases slightly up to term (5, 6). In 30 normal pregnant women followed with serial vital capacity

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determinations in our laboratory, we found no consistent evidence of either an increase or decrease during pregnancy. Under the influence of aldosterone and other hormones, sodium is retained and total body water increases up to term.

From these physiological considerations, it is apparent that the peak cardiovascular load is reached somewhere in the seventh month and that the hemodynamic burden progressively lightens from then until the time of delivery. The clinical corollary of this is that if the cardiac patient successfully weathers the seventh month, she is unlikely to encounter difficulties thereafter. Labor, which represents intermittent muscular exertion, is apparently better tolerated than the earlier sustained hemodynamic burdens in the sixth and seventh months.

In the first 24 hours postpartum, abrupt vascular changes develop. An increased venous return from the uterus and the legs is translated into an increasing cardiac output from an average of 5.5 to 7.1 liters (29 per cent) (4). At this time the vital capacity abruptly falls 200-300 ml. (7)

PATHOPHYSIOLOGY

RHEUMATIC DISEASE accounts for at least 90 per cent of the cardiovascular lesions in pregnant women; of these, about three-fourths consist of mitral stenosis alone or in combination with other lesions. Our comments will be confined to the care of rheumatic patients and will concern mitral stenosis in particular, although most of what is said in subsequent sections obviously will apply to any form of heart disease.

When the burdens of heart disease fall upon the young left ventricle, as in aortic stenosis or mitral regurgitation, this chamber has amazing powers of adaptability and compensation through hypertrophy and increased working power. The pregnant cardiac, however, much to her sorrow, usually has neither of these lesions alone; instead, mitral stenosis predominates so that the burden is imposed not on the thick-walled left ventricle but on the thin-walled left atrium and right ventricle. These chambers must deliver their quota of blood through an obstructed outlet to the waiting left ventricle, which necessitates increasing the normal gradient across the mitral valve. Because of the increased heart rate and increased cardiac output of pregnancy, the pregnant patient must transfer more blood in a shorter time through the obstructed valve, and the gradient must therefore increase further. The resulting high left atrial pressure is shared by the pulmonary veins and capillaries; and in severe mitral stenosis, the pregnant patient can never be far from the brink of pulmonary edema (or hemoptysis). As pulmonary hypertension progresses, changes in the pulmonary arterial bed afford some measure of protection against the influx of arterial blood and so against pulmonary edema (and hemoptysis); at this later stage in the natural history of mitral stenosis, the threat of pulmonary edema is less, but the increased burden on the right ventricle leads to right-sided failure with peripheral congestion and edema.

INITIAL CLINICAL EVALUATION

AT THE INITIAL evaluation, the pregnant cardiac can generally be recognized as a "good risk" or a "poor risk." Far and away the most important single point in assessment is her exercise tolerance—if she can run up two flights of steps without breathlessness, she will probably experience no difficulties in childbearing. There are exceptions to this general rule; we have seen one mother, asymptomatic before pregnancy, who developed brisk hemoptysis in her second trimester and pulmonary edema in her seventh month and again during labor. Since delivery she has once more been completely free of all symptoms.

The "best risk" patient has no symptoms, a normal-sized heart, and mitral insufficiency as her only valvular defect. Gorenberg (8) reports no cases of congestive heart failure in patients with mitral insufficiency alone. Most of such patients would probably get through their pregnancy satisfactorily without any special medical care. Apart from this favorable situation, there is probably little prognostic value in the type of valvular lesion, although Ullery (2) states that the prognosis is worse with a double valvular lesion; he reports seven per cent mortality in patients with combined mitral and aortic lesions as against 1.9 per cent in those with mitral involvement alone. Patients with additional lesions besides mitral insufficiency will generally do well with proper medical care, but if not properly supervised, they are in danger of becoming "bad

risks." The "worst risk" category comprises those who have had symptoms of cardiac insufficiency before their pregnancy.

Other factors, besides functional class and type of valvular lesion, that enter into the initial evaluation include age of patient, size of heart, disturbances of rhythm, and concomitant diseases. Other things being equal, the rheumatic patient of 38 has less cardiac reserve than does her sister of 24; and an obviously enlarged heart possesses less reserve than a normal-sized or only slightly enlarged one. Atrial fibrillation adds greatly to the likelihood of failure and causes an abrupt increase in mortality. Of 121 fibrillators compiled from several series of patients between 1921 and 1953, 42 (35 per cent) died (9, 10, 11). With modern methods of management, this mortality should be considerably reduced; but atrial fibrillation remains a serious threat to the childbearing rheumatic.

At the initial visit, it is vitally important to secure the proper cooperation of the patient and to explain to her in reassuring terms the realities of her situation. She should be told that if she cooperates to the utmost, she has an excellent chance of passing safely and uneventfully through pregnancy and delivery. It should be explained to her that her heart's reserve, subnormal at the best of times, is further compromised by the hemodynamic burden of pregnancy; therefore, she must not hesitate to seek help if any additional stress (such as a respiratory infection) befalls her. The role of *infection* must be stressed and stressed again. Vander Veer (1) reported that of 11 fatal cases of congestive heart failure in pregnancy, seven were precipitated by respiratory infections. If the patient is not on prophylactic penicillin, she should be put on adequate dosage at once, not only as a preventative of rheumatic recurrence but also to reduce the incidence of respiratory infections, that might fatally tip the scales.

Her entire regimen should be outlined, including instructions concerning *diet* and *rest*. Every cardiac patient should have at least 10 hours rest at night and should also rest, in whatever position is most comfortable to her, for at least an hour or two every afternoon. *Physical activity* must be appropriately restrained. The doctor, his patient, and her family should thoroughly appreciate the implications of *emotional upsets,* since an emotional storm can greatly increase the output of the heart and, with it, the cardiac work.

Although the frequency of her visits will depend on the initial evaluation and on her subsequent progress and will vary from monthly visits in the mildest cardiacs with only mitral insufficiency to visits as often as once or twice a week, she should understand clearly that if she has the slightest hint of deterioration, she must immediately get in touch with her medical advisor. She must be instructed to report any new symptoms; special emphasis must be placed on infections, shortness of breath, undue fatigue, swelling of the feet, gain in weight, and palpitations. Hemoptysis is so dramatic that it is unlikely to go unreported.

FOLLOW-UP VISITS

At subsequent follow-up visits, routine inquiry should be made concerning such obviously cardiac complaints as shortness of breath, swelling of the feet and ankles, and hemoptysis. Other less obvious danger signals include cough, undue fatigue, insomnia, and nightmares. Above all, the importance of infections, even minor ones, should be stressed by never failing to inquire about them.

On physical examination, the weight and blood pressure are routinely recorded, as are the heart rate and rhythm and the respiratory rate. A heart rate of more than 90 in a patient with significant mitral stenosis should be treated with considerable respect; over 100 it is a danger signal, and over 110 it must be regarded as a potential emergency and treated accordingly with strict rest. Edema of the feet and ankles, venous congestion in the neck, rales at the lung bases, and enlargement of the liver are regularly looked for.

It is important to realize that many of the physical signs of heart failure can be closely imitated by the hyperdynamic circulation of pregnancy. Edema, moderate tachycardia, palpitations, dyspnea, and unusual fullness of the neck veins are common accompaniments of normal pregnancy. The heart may appear enlarged, both because of the higher diaphragm and, perhaps, because there is actual physiological enlargement. An accentuated pulmonic component to the second sound is also a common physiologic finding. Transient atelectatic rales at the lung bases disappear-

ing after a few deep breaths are also common during pregnancy, but persistent rales are *not* physiologic.

The distinction, therefore, between early congestive failure and the physiologic state may be difficult to make. In these circumstances, two tests may prove helpful: the vital capacity and a therapeutic test of diuretics. A decreased vital capacity may signal early pulmonary congestion even before subjective dyspnea develops. When dyspnea is already present, if it is due to the hyperventilation of normal pregnancy, there should be no decrease in vital capacity; whereas if it is due to pulmonary congestion, it should be attended by a definite reduction in vital capacity. In the presence of congestive failure, the administration of a potent diuretic should produce an improvement in symptoms together with a loss of several pounds of body weight; whereas the normal pregnant patient should obtain no subjective relief and lose relatively little fluid and weight, two or three pounds at the most. In doubtful cases, a prolonged circulation time may help to establish the diagnosis of congestive failure.

In the pregnant cardiac clinic at the Mercy Hospital, the vital capacity is routinely obtained at each visit, and any decline of more than 200 ml. is viewed with grave suspicion of impending congestive failure. A decreasing vital capacity has been found (5, 6) to be an excellent early index of pulmonary congestion. Provided the tests are performed with a cooperative patient in the same position with the same spirometer and preferably by the same technician, changes of 200-300 ml. are definitely significant. The hemoglobin should be checked at least monthly throughout the pregnancy; an unsuspected anemia can steal dangerously from the heart's reserves.

PRACTICAL MANAGEMENT

THE PRIMARY GOAL in the management of the pregnant cardiac is the prevention of heart failure. It is wise to approach her with the attitude that there is only one disaster that can befall her—congestive heart failure. Every minor malady must be treated as if it were an equivalent of heart failure; for example, a mild respiratory infection should be treated with strict rest. Frequent visits ensure that the earliest signs of failure will be recognized and that the patient will better be kept toeing the line. In collaboration with her obstetrician, she should be maintained on an appropriately restricted sodium intake, and her weight gain should be limited. If she is overweight at the outset, she should be put on a low calorie diet and should be encouraged to lose flesh. There is no contraindication to a reducing diet during pregnancy; indeed, it is clearly indicated for the fat patient, especially if she starts with a cardiac handicap.

If, despite careful surveillance, she develops congestive failure, the treatment is no different from that of any other type of cardiac in failure; but the stakes are higher. Digitalization should be initiated, and it is much better to err on the side of premature use; far better to digitalize a patient who does not need it, than not to use the drug when failure is incipient. We emphasized before that the assessment of symptoms and signs during pregnancy may be extremely difficult; we believe that if there is any doubt, treatment for congestive failure should be instituted. If the heart rate at successive visits creeps upwards faster than it should, it may be wise to initiate digitalis. If there is an unexpected gain in weight, or if an unproductive cough develops, or if the patient's breathlessness or orthopnea seem to be increasing more than we expect during a normal pregnancy, or if the vital capacity inexplicably falls by 200-300 ml., it is probably wise to institute diuretic therapy with or without digitalization. The potent oral diuretics now available are a great boon to the failing pregnant cardiac.

It has been repeatedly stressed that patients who go into heart failure during pregnancy should be hospitalized and kept at bedrest until delivery. This is a counsel of theoretical perfection. It is completely true that cardiacs who develop failure should have appropriate rest until after delivery, but neither the hospital nor the bed provides any guarantee of proper rest. In this day of fantastic hospital costs, the financial burden of several weeks or months of hospitalization and its attendant mental anguish may more than outweigh the advantages. The decision whether to admit the patient to a hospital rests on many factors: the home circumstances, willingness and ability of the patient and family to follow instructions faithfully at home, the financial status of the family, ability of the physician to visit the patient in her home, and so forth. Secondly, bed is

no place for any cardiac except for siestas and sleeping, and even for these a comfortable chair is often to be preferred. Admittedly, "bedrest" is a cliché that does not always mean precisely what it says, and through unfortunate usage it has come to be synonymous with maximal rest. The evils of too much time in bed, however, cannot be overemphasized in the handling of cardiacs of all sorts; and the pregnant cardiac is no exception.

Many physicians have by now learned the lesson that surgical interference in the form of therapeutic abortion or cesarean section is never indicated on cardiological grounds (8, 12, 13). On the other hand, it is now becoming fashionable to consider mitral valvotomy as a desirable procedure during pregnancy. It may well be that a few individual patients exist in whom such a procedure may justifiably be recommended, but these are few and far between. As a general rule, it is safe to say that such surgery, if indicated, should be postponed until after delivery. There are many reasons for postponement: although it is true that pregnant rheumatics tolerate valvotomy well enough, a definite mortality is associated with the operation (as much as 11 per cent has been reported (14)); and every time a mother is lost, her baby goes too. This double mortality makes the operation all but prohibitive, especially when it is appreciated that, with careful medical management, the overall mortality of pregnant cardiacs today is about 1 per cent. Burwell and Metcalfe (13) have yet to recommend a valvotomy during pregnancy. Metcalfe (15) quotes the following figures in support of his reluctance to recommend mitral surgery during pregnancy: of 300 patients followed conservatively by themselves, only three died; whereas, of 14 patients in other hands who were submitted to commissurotomy during pregnancy, four died. To further illustrate that commissurotomy is no sure answer to the cardiac burdens of pregnancy, he draws attention to four patients who, having survived a "successful" valvotomy when not pregnant, died during their next pregnancy. Nevertheless, it is possible that an occasional carefully selected patient should have her mitral obstruction relieved surgically during pregnancy. In these exceptional cases, the operation should be performed in the first trimester, in order to derive the full benefit of the surgery by the time the patient approaches the period of maximal hemodynamic load in the third trimester. Occasional dire emergencies may demand that the operation be performed later (16, 17).

It is wise to admit all but the mildest cardiacs to the hospital a week or two before delivery to avoid the stress of a rushed admission, to allow the patient to become acclimatized to the hospital atmosphere and establish rapport with her attendants, and to ensure that she is in the optimal condition for the stresses of labor and delivery.

The relationship of toxemia to heart disease has not been clearly defined, but there is at least suggestive evidence that the presence of heart disease predisposes to the development of preeclampsia. For example, Burt (18) found that whereas only 2.6 per cent of 717 patients with toxemia had heart disease, 19 of 52 (36.5 per cent) patients with heart disease developed toxemia. We have no sure way of staving off the development of toxemia, but by realizing its proclivity for attacking the cardiac and the extreme seriousness of the added burden if it does, we can be on the lookout for the first signs of it and then act vigorously to modify its effects.

To summarize: the prophylactic management of the pregnant cardiac has two aims: (1) to minimize the unavoidable hemodynamic burdens of the pregnant state, and (2) to fend off avoidable stresses and strains. The physiologic burdens of pregnancy may be outlined as follows: salt and water retention, increased blood volume, increased cardiac output, increased heart rate, and increased weight. As these loads cannot be avoided, good management strives to minimize them. Sodium is restricted, diuretics are used, exertions—physical and emotional—that increase cardiac output and heart rate are avoided, digitalis and rauwolfia are employed, when indicated, for their bradycrotic effect, and weight gain is held to a minimum consistent with good general health. At the same time, a real effort is made to protect the patient from avoidable or partially avoidable hazards, such as infection, anemia, and toxemia.

MANAGEMENT DURING LABOR AND POSTPARTUM

PROVIDED THE PREGNANCY has been properly supervised and heart failure has not previously developed, there is usually nothing to fear from the intermittent muscular exertions of labor. But

vigilance must not be relaxed. The patient should be adequately sedated, and pulse and respiratory rates must be repeatedly checked; any rise toward 110 and 24, respectively, should be regarded as evidence of impending heart failure. Atropine and scopolamine are better avoided because of their tachycrotic effect. It is safer to keep the patient out of bed and sitting in a chair for as much of the first stage as possible. We have seen a patient with pure mitral stenosis develop, during the first stage of labor, pulmonary edema, which was promptly reversed by the simple expedient of getting her out of bed and sitting her in a chair. If the patient is in bed, the head of the bed should be raised as high as is practicable; but this does not compensate for the advantages of keeping the legs dependent.

Again, on the delivery table, the patient's head should be kept unconventionally high; and the Trendelenburg position obviously must be avoided. Some form of spinal anesthetic, which encourages pooling of venous blood in the legs, is often recommended. We have found saddle-block, using no more than 3 mg. of Pontocaine,® to be satisfactory. Low forceps are usually recommended in order to obviate any undue prolongation of the second stage. We would emphasize that natural delivery is much to be preferred; indeed, there are no cardiological indications for cesarean section.

Immediately postpartum, the vital capacity falls abruptly, and the venous return and cardiac output rise; this, therefore, is a danger period. We have recently observed a patient with no cardiac symptoms or history, whose mitral stenosis went undetected throughout pregnancy, develop pulmonary edema within a few hours of delivery. Of 126 fatal cases of congestive failure associated with childbearing, four developed for the first time postpartum (19). It is, therefore, important to minimize the readjustments occurring at this time. Some authorities apply venous tourniquets to the legs; the head should be kept high, and the already increased venous return should not be augmented by giving oxytocics if these can be avoided. Parenteral fluids should be avoided or kept to a minimum, and no sodium should be included in them. Moderate hemorrhages, provided they have been controlled, should *not* be corrected by blood transfusion. Penicillin and streptomycin are given during the third stage and are continued for three days postpartum, to forestall bacterial endocarditis.

Throughout labor and delivery, emergency equipment should be at hand, including at least the following: tourniquets, donor set, morphine, quinidine, Pronestyl,®Strophanthin® or ouabain, intravenous Cedilanid® or digoxin, oxygen (possibly with equipment for administration under pressure or for bubbling through alcohol).

Salt restriction and rest are carefully maintained in the first two weeks postpartum. No measures which have been found necessary during the pregnancy (e.g., digitalis, diuretics) are withdrawn for at least ten days. It is often advised that the patient be kept in bed for the first week or so and not be discharged from the hospital for a fortnight. Once again the same considerations apply as applied to bed rest and hospitalization during pregnancy. There is no question that cardiacs postpartum should be kept under close medical observation for a longer span than their normal sisters, but that they should remain in bed is absurd. If it is wise obstetrically to sit the normal mother up early, there is all the more reason for giving the cardiac patient the benefit of sitting in a chair with the feet dependent. This is not to be interpreted as a license for additional activity; her rest should be maximal, which means that she must be capably assisted, if not bodily lifted, out of bed and into the chair. The time of discharge can be determined individually; in our opinion, any mild cardiac who has passed through pregnancy, delivery, and the puerperium without mishap may certainly be discharged at the end of a week if home arrangements are suitable.

If sterilization has been decided upon (a consideration of the socio-religious aspects of this question is beyond the scope of this brief paper), it should be postponed until at least two weeks postpartum (20). If one can be sure that the patient will return before she becomes pregnant again, it is probably better to readmit her for this procedure six or eight weeks later (21).

There is no cardiological reason why the mother who has successfully accomplished pregnancy and delivery should not breast-feed her hard-won infant. Indeed, there is much to be said for the philosophy that both delivery and nurture of the baby should be *per vias naturales.*

In the nineteenth century, the mortality rate

for cardiacs who undertook to bear children was almost 50 per cent. Through the 1940's, reported mortality rates still ran between 3 and 4 per cent. By 1955, however, death rates had dropped to less than 1 per cent; in several series totaling well over four thousand cardiacs (1, 8, 21-23), there were but 33 deaths. This is indeed cause for congratulation, but not for complacency.

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Book Reviews

Blood Diseases of Infancy and Childhood, Carl H. Smith, M.D., St. Louis, The C. V. Mosby Company, 1960.

This book presents the essentials of pediatric hematology in concise form for the medical student and practitioner. It is the first book to provide the salient features of blood dyscrasias against the background of normal development of infancy and childhood and is a worthwhile addition to your medical library.

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Infectious Diseases of Children, Saul Krugman, M.D., and Robert Ward, M. D., St. Louis, The C. V. Mosby Company, 1960.

The rapid accumulation of knowledge of viruses and of human diseases caused by them is chiefly responsible for this endeavor—a revision of the original book published about two years ago. Its purpose is to provide a concise and handy description of certain common infectious diseases of children. It is written primarily for pediatricians, general practitioners, and medical students who deal with children.

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Resuscitation of the Newborn Infant, Harold Abramson, M.D., editor, St. Louis, The C. V. Mosby Company, 1960.

This is the story of the resuscitation of newborn infants—what is known and what is not known. It describes a problem as old as mankind, yet a problem not satisfactorily solved.

The opinions advanced by no means represent the final word. As experimental and clinical research reveal appropriate answers to the many unexplained phases of the problem, changes must necessarily ensue in the clinician's approach to the everyday application of methods of resuscitating newborn infants.

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A System of Medical Hypnosis, Ainslie Mears, M.D., Philadelphia, W. B. Saunders Company, 1960.

The author, who is president of the International Society for Clinical and Experimental Hypnosis, describes medical hypnosis as he practices it. He makes no claims to revealing anything new, but relates the manner in which he uses medical hypnosis and his reasons why, viewing each individual maneuver and each particular procedure as part of the overall method.

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Complications in Surgery and Their Management, Curtis P. Artz, M.D., and James D. Hardy, M.D., Philadelphia, W. B. Saunders Company, 1960.

The first part of this volume is concerned with general complications pertinent to all operations; the latter part is a more definitive treatise on specific operations of various anatomic areas. A chapter has been devoted to complications of anesthesia. It is anticipated that this volume will bring to the attention of surgeons, residents, and students the techniques and warnings of nationally recognized authorities in various phases of surgery.

A description of the clinical features, diagnosis, and treatment of

PULMONARY HYPERTENSION AND COR PULMONALE

Robert C. Duvall, M.D.

Pulmonary Hypertension

IN THE UNEXPANDED, unventilated fetal lung, the pulmonary arteries and arterioles appear thick walled, constricted, and kinked, with well-developed muscular layers. This arrangement produces a high pulmonary vascular resistance and virtually no flow of blood during fetal life. The histology of these vessels is similar to that of the vessels in the systemic circuit. The large pulmonary arteries have an aorta-like configuration with long, closely packed elastic fibers. Within three months after birth, these hypertrophied pulmonary arteries undergo involution with change in the elastic fibers in the direction of their becoming short, broken up, and relatively widely spaced.

Upon expansion of the lungs at birth, the pulmonary vascular resistance falls to systemic levels; within a few hours, it falls even further An aorto-pulmonic shunt through the ductus ensues till this structure closes, at which point pulmonary artery pressure declines further; vasoconstrictor tone is released; and structural involution is encouraged so that the normal adult anatomical and physiological arrangement is established at the end of the third month of life.

Pulmonary hypertension is usually defined as that level of pressure existent above 30 mm. of mercury, systolic, and 15 mm. of mercury, diastolic. Practically speaking, levels above 50/25 might be more realistic upper levels of normality, as pressure levels up to these generally cause no adverse clinical effects.

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Patho-physiologically, pulmonary hypertension is classified as follows:

I. *Passive pulmonary hypertension* is exemplified by high pulmonary venous pressure from whatever cause, including left ventricular failure in hypertensive cardiovascular disease, mitral stenosis in rheumatic heart disease, myxomata of the left atrium, and multiple pulmonary venous thromboses. Physiologically, these lesions cause a decrease in cardiac output and an increase in pulmonary artery pressure due to high pressure in the left atrium or pulmonary venous system. The pulmonary arterial hypertension so produced rarely influences the fundamental disease causing it; rather the findings are those that result from high pulmonary venous pressure.

II. *Hyperkinetic pulmonary hypertension* results from increased pulmonary blood flow. Riley and others have established the fact that normal individuals tolerate increased blood flow to at least the level of three times the resting cardiac output without any concurrent increase in pulmonary artery pressure, presumably because of concomitant fall in pulmonary vascular resistance, whether through opening of new pulmonary vascular channels or in further widening of those vessels already open. There comes a point, however, when the vascular bed is fully open in the presence of high blood flow and a relatively slight increase in pulmonary vascular resistance may produce a rather marked increase in pulmonary artery pressure. Examples of lesions producing this type of situation include atrial and ventricular septal defects, ductus arteriosus. Reactive change in the pulmonary vascular tree (vasoconstrictor

tone or arteriosclerosis of the arterioles) may worsen this situation.

III. *Vaso-occlusive pulmonary hypertension* is the type of pulmonary artery hypertension caused by loss of vascular patency at the capillary, arteriolar, or arterial level as a result of several pathologic mechanisms:

A. Obstructive lesions that block blood flow from extrinsic vascular compression or intrinsic occlusion; for example, multiple or recurrent pulmonary emboli. When these lesions are of long standing they may induce "reactive" structural changes as described under hyperkinetic pulmonary hypertension.

Weidner and Light, in dog studies, found that emboli measuring 25 to 10 microns in diameter, when injected in segmental pulmonary arteries or in generalized distribution, produced pulmonary artery hypertension, whereas larger particles failed to do so. This was true when both large and small particles were given in modest doses. After sympathectomy, in this experiment, generalized embolization with small particles produced pulmonary hypertension only after a massive dose had been administered. Presumably this reflected actual reduction in available vascular space and no longer vasoconstrictor influence arising through stimulation of a specific area of the vascular tree.

B. Obliterative pulmonary hypertension resulting from a decreased vascular capacity through disease of the vessels themselves; i.e. various blood dyscrasias, lupus, and other arteritides, schistosoma infestation, and, most significantly, emphysema and diffuse interstitial fibrosis. In the last two entities, in addition to a simple mechanical change, a mixture of vasoconstrictor influence and hyperkinesis probably enters into the production of pulmonary artery hypertension.

C. Vasoconstrictive pulmonary artery hypertension resulting from functional contraction of the arteries, as in "primary" pulmonary artery hypertension, bronchitis, and asthma. In the last two entities, it is assumed that hypoxia stimulates vasoconstrictor tone, thus leading to transient pulmonary artery hypertension.

Primary pulmonary hypertension has aroused some interest in the past decade. It is an illness of unknown cause that predominates in females over males in a ratio of 5 to 1. This last fact militates against its being regarded as a thrombo-embolic phenomenon. It probably represents some overactivity on the part of the sympathetic nervous system, for there occurs in this disease a greater fall in pulmonary artery pressure as a result of injection of acetyl choline than in any other form of pulmonary artery hypertension. Priscoline® also produces a marked effect.

The disease generally produces no symptoms until it is fully developed; then it manifests itself by limited effort tolerance, fatigue, breathlessness, "anginoid" distress, and faintness. The signs include low blood pressure, small pulse pressure, split loud P-2, and, sometimes, pulmonary diastolic murmur. The physiologic findings include high P.A. pressure, high right ventricular enddiastolic pressure, reduced cardiac output, and increased A-V oxygen difference with normal arterial saturation till right heart failure ensues. Failure and death usually occur within two or three years of onset the symptoms in patients who receive no therapy. Current treatment with anti-coagulants, reserpine, Priscoline®, and aminophylline have prolonged the life span.

The small pulmonary arteries are thought to be the site of the increased resistance, although Dresdale, in his review a few years ago, noted the great variability in the nature, severity, and distribution of pulmonary vascular changes.

Cor Pulmonale

THE CONSEQUENCE of the hypertension that ensues from the foregoing lesions and mechanisms is *cor pulmonale,* a term first used by Paul White in 1931. As did most other writers in this field, White excluded increased pulmonary vascular resistance due to mitral stenosis, left heart failure, or congenital heart disease.

Cor pulmonale is hypertrophy of the right ventricle, with or without failure, caused by increased pulmonary vascular resistance. In considering diseases of the lung which are responsible for cor pulmonale, restriction of the vascular bed is a more important cause of right heart strain than is increased flow. Even patients with a pneumonectomy can tolerate flow two to three times normal with minimal rises in pulmonary artery pressure of the remaining lung is free of fibrosis, emphysema, or vascular change. This observation was reported by Cournand in 1950. More recently, Brofman, working with human

subjects and occluding one pulmonary artery with a balloon-type cardiac catheter, demonstrated that increased flow of eight to ten times normal is tolerated with minimal rise in P.A. pressure.

The incidence of cor pulmonale is vaguely documented. Postmortem studies more than 20 years ago suggested that selective right ventricular enlargement in emphysema is uncommon. McKeown, however, in doing autopsies on 101 cases of emphysema, in 1952, found right ventricular enlargement in 40 per cent. Other autopsy series have suggested that in most cases with pulmonary disease, the right ventricle is normal and that little correlation exists between right ventricular thickness and pulmonary vascular changes except in the more severe cases. Certainly, the vast majority of patients with pulmonary tuberculosis, even advanced cases, do not show cor pulmonale until or unless their disease remains extant for many years and becomes complicated with obstructive emphysema. Emphysema is our great problem in the field of respiratory disease today. Many of the victims of this illness must have had right ventricular hypertrophy when the disease had been present for many years, but their deaths have probably been ascribed to other causes, most frequently pneumonia.

One classification of cor pulmonale follows:

I. *Cor pulmonale in cases of pulmonary disease with chronic obstructive emphysema*—Treatment of these diseases is usually more satisfactory than is true of cor pulmonale in diseases involving pulmonary vessels. Chronic diffuse obstructive emphysema is the commonest disease encountered in this class, and the commonest cause of this entity is chronic bronchitis. Asthma is associated with chronic cor pulmonale only if severe, of long duration, and associated with secondary infectious bronchitis. Kypho-scoliosis is associated with this class of disease through recurrent infections and subsequent production of obstructive emphysema. Tuberculosis, when long standing, may be associated with chronic obstructive emphysema and, thereby, ultimately with cor pulmonale. Sarcoidosis may manifest itself as a diffuse peribronchiolar granuloma with the production of obstructive emphysema.

The pathogenesis of cor pulmonale from pulmonary diseases associated with chronic obstructive emphysema involves the following factors:

A. Increased resistance to blood flow. Among the mechanisms involved here are:

1. Reduced cross sectional diameter and distensibility of the vascular bed as a result of ruptured septa with loss of capillaries and compression of others by bullae; areas of scarring; compression of vessels from scattered alveolar exudate and atelectasis; cuffing of blood vessels by inflammatory cells; direct action of hypoxia on vessels. (The exact site of action of this last named mechanism is not clear. There is some evidence that, in the human, hypoxia produces an increase in pulmonary artery pressure but not an increase in wedge pressure.)

2. Polycythemia, which, if present, leads to hypervolemia from an increase in red cell mass. This, in turn, is associated with increased venous return to the heart, increased cardiac output, and an increased residual volume of blood in the lungs. Such an attempt to compensate for arterial desaturation by providing the tissues with extra amounts of oxygen per unit volume of blood and per unit time ultimately works to the disadvantage of the host through heart failure.

3. Intrapulmonary vascular shunts of various sorts, which contribute to the development of cor pulmonale in these cases:

(a) Precapillary bronchial arterial to pulmonary arterial shunts. Their presence has been shown in the diseased lung, although not so clearly in the normal lung. The effect of this is to increase the blood flow in a lung which already has a restricted vascular bed, and, therefore, to further contribute to increases in pulmonary artery pressure. Such shunts must also increase left ventricular work. In bronchiectasis, evidence indicates that there may be reversal of flow in the pulmonary artery toward the hilum. Such a mechanism might shunt desaturated pulmonary artery blood away from poorly ventilated areas of lung.

(b) Venous anastomoses. Bronchial vein to pulmonary vein anastomoses are known to exist in the normal. The bronchial veins drain only the large bronchi. Flow is usually from the pulmonary veins to bronchial veins, since the left atrial pressure is somewhat higher than the right and there are valves to encourage flow in this direction at the junction of the bronchial veins and the azygos systems. In emphysema, expansion of these communications occurs and may cause

reversed flow in these anastomoses in some situations; e.g., when high azygos pressure in heart failure is accompanied by incompetence of the above valves, the venous blood could conceivably flow through these venous anastomoses into the systemic arterial circuit. The full significance of the above bronchial arterial and bronchial venous shunts in emphysema is uncertain, however.

B. Cardiac output. Is this increased or decreased in these disease states? This depends on the type and extent of pulmonary disease—the presence of bronchitis or pneumonitis, the degree of hypoxia, the presence of polycythemia, the size of the blood volume, and the presence or absence of failure.

1. Mechanisms which increase cardiac output: These include hypoxia, polycythemia and hypervolemia, infection with its resultant increase in oxygen consumption and increase in cardiac output; increased work of breathing with resultant increases in oxygen consumption and output. If cardiac failure occurs, cardiac output may fall but still remain above normal values. With failure, the increase in plasma volume helps to maintain cardiac output, but later, with over distention of the right ventricle, may lower the output.

2. Factors which produce low cardiac output: This is true in severe failure. If the factors serving to increase cardiac output cannot overcome an even higher pulmonary vascular resistance, the cardiac output will be low.

II. *Cor pulmonale in cases of pulmonary disease involving the pulmonary vasculature*—These diseases may be intra-luminal, as exemplified by diseases associated with pulmonary emboli, primary pulmonary hypertension, sickle cell disease, and schistosoma parasitic infestation; or they may be extra-luminal, as in pulmonary interstitial fibrosis or granulomata, scleroderma, Hamman-Rich syndrome, diffuse pulmonary carcinomatosis, compression of the main pulmonary arteries by metastatic tumor, lymph nodes, and aneurysm.

The pathogenesis of cor pulmonale associated with the above diseases involves a reduction in vascular caliber or actual loss of capillary bed. Initially, hypoxemia occurs at exercise only, but later it occurs at rest as well. Polycythemia is sometimes present. The cardiac output in the diffuse granulomatoses tends to be high. The reason for this is not certain. Perhaps active disease results in increased metabolism and associated increased oxygen consumption, or perhaps the increased work of breathing is the common denominator; however, with desaturation and failure, cardiac output drops. In diseases associated with pulmonary emboli and primary pulmonary hypertension, the cardiac output tends to be low.

Both types of cor pulmonale may be present in such diseases as silicosis and sarcoidosis.

Diagnosis

DIAGNOSIS OF COR PULMONALE rests upon recognition of the background disease that may alter the pulmonary circulation. Superimposed at some time in the course of such a disease are the symptoms of pulmonary hypertension itself, which include fatigue, dyspnea, cyanosis, "anginoid" pain, and, if the cardiac output is low, dizziness and syncope.

The physical signs include dyspnea, cyanosis, perhaps clubbing, apprehension, possibly some cardiac enlargement, lower or left parasternal thrust, accentuated P-2, perhaps a diastolic shock; systolic and diastolic murmurs in the pulmonic area are common. Interestingly, hydrothorax caused by right heart disease is rare, and arrhythmias are unusual.

X-ray findings include a prominent artery segment and variable pulmonary vascular markings. The latter are increased in mitral stenosis and atrial septal defects but reduced in primary pulmonary vascular sclerosis and primary pulmonary hypertension. The heart may not appear enlarged on the P.A. film, or it may sometimes be mistakenly regarded as showing left ventricular enlargement when clockwise rotation occurs.

The EKG pattern may be normal in as many as 30 per cent of the cases of cor pulmonale. Since the classic EKG findings of right heart disease are well known, they will not be described here.

Treatment

COR PULMONALE TYPE I—Many of the changes in chronic obstructive emphysema are reversible, and the prospect is good for improvement as a result of treatment. Principles include:

1. Combating infection with antibiotics.
2. Maintaining airway with bronchodilators,

with bronchial aspiration, breathing exercises, perhaps steroids for edema, secretions, and spasm; even tracheostomy may be necessary.

3. Treating hypoxia with an awareness of the dangers of carbon dioxide retention and subsequent narcosis. It is well to use sedatives cautiously in such individuals for fear of further depressing respiration. Various positive pressure breathing devices have been popular in recent years, the Bennett and Emerson respirators being the ones most widely used. These devices may be useful in the acute illness and, when tolerated by the patient, serve well to increase alveolar ventilation. They must generally be used in association with some moistening agent or bronchodilator, such as Alevaire® or Isuprel®.

4. Maintaining a cardiac regimen. In spite of earlier doubts about its usefulness, virtually all workers in this field now favor the use of digitalis in cor pulmonale and failure; however, the heart rate is a poor index of the degree of digitalization in these individuals. The other principles of cardiac therapy apply, including low salt diet, rest, and diuretics. Phlebotomy is often employed in individuals whose hematocrit exceeds 55.

5. Using ganglionic blocking agents in emphysema to combat vasoconstrictor influence is largely an unexplored matter.

Two complications of which to beware in emphysema are peptic ulcer, a common accompaniment of emphysema, and cholelithiasis, which sometimes occurs in cases with secondary polycythemia.

COR PULMONALE TYPE II—Many of the foregoing principles also apply in treatment of these cases. When types I and II are mixed, as in complicated silicosis and sarcoidosis, one treats the infection and the emphysema. In those instances of granulomatous replacement and compression of vascular beds, steroids have been used in recent years with varying results. Unless steroid therapy is contraindicated, their use is probably to be encouraged in these illnesses in which the outlook is so grave and other therapies so useless. When cardiac failure ensues, exodus is generally rapid as a result of irreversible high pulmonary vascular resistance.

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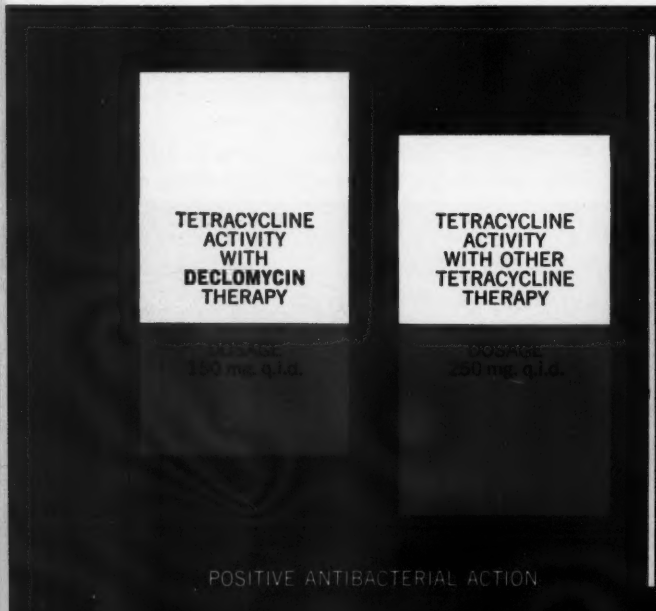

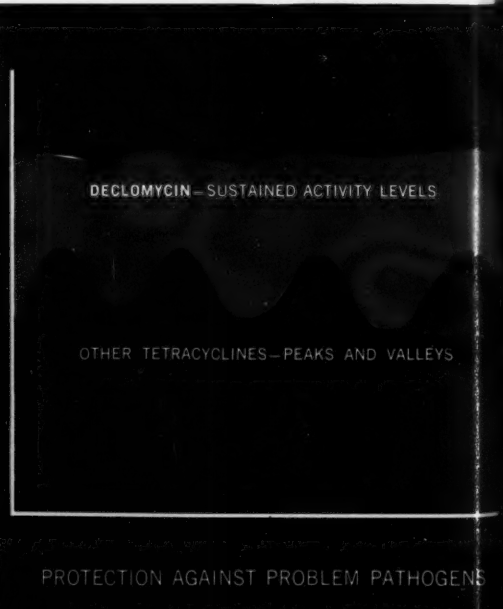

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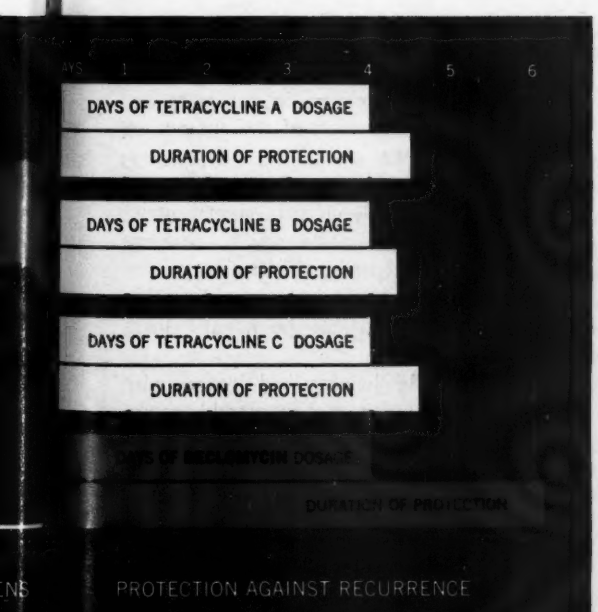

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REPORT OF A PATIENT WITH PRIMARY PULMONARY HYPERTENSION

A 32-year-old white woman was admitted for the first time to The Johns Hopkins Hospital on February 3, 1957. Her chief complaints were shortness of breath and "passing out spells for the past two years."

Past history revealed that the patient had enjoyed excellent health until two years before admission. She had one bout of jaundice at the age of 20 and had experienced no difficulty with her first pregnancy, seven years before. During her second pregnancy, exertional dyspnea appeared; and, in the latter months, she also had peripheral edema and ascites. Her first syncopal episode occurred during the second month of this pregnancy. Five days after discharge from the hospital, she had her second syncopal episode, which was much more severe since she did not regain complete consciousness for 45 minutes. No associated incontinence or convulsive movements had occured with either episode. She delivered her second child on July 8, 1955.

On November 1, 1955, cardiac catheterization was done at the Allegheny General Hospital, Pittsburgh, Pennsylvania. The interpretation read, "Severe pulmonary artery hypertension with mild right ventricular failure. The cause is not apparent." The pressures in millimeters of mercury in the pulmonary artery were 111/42-69, and in the right ventricle, 111/8-14. Cardiac index was 1.8 liters per minute; cardiac output was 3.1 liters per minute. In November 1955, diuretic therapy was begun, and digitalis was started one month after delivery of her second child. Hepatomegaly and peripheral edema had increased three weeks before the first admission to Johns Hopkins Hospital. The last syncopal episode took place one month before admission. They had been occurring at intervals of one to two weeks.

Physical examination on admission revealed the pulse to be 70 per minute and the blood pressure 100/80 in the right arm. The patient was well developed, well nourished, and in no acute distress. Her body configuration was normal, and there was no clubbing or cyanosis. Her lungs were clear to percussion and auscultation.

Definite slight neck vein distention was noted in the semi-recumbent position, and a prominent right ventricular precordial heave was present. The heart was enlarged slightly to the right in the third and fourth right intercostal spaces. There was increased dullness in the second and third left intercostal spaces; the left cardiac dullness extended to midway between the left midclavicular and left anterior axillary lines. A palpable systolic thrill was felt maximally in the fourth and fifth left intercostal spaces along the left sternal border.

On auscultation, the heart sounds were of good quality with regular sinus rhythm. The second pulmonic sound was greatly accentuated, and a high pitched, blowing diastolic murmur was heard along the left sternal border from the second to the fourth interspaces. A grade IV, harsh, blowing systolic murmur was heard maximally over the body of the sternum and in the third, fourth, and fifth left intercostal spaces, varying with the phase of respiration and being maximal at the height of expiration. No opening snap of the mitral valve was heard, nor was the first mitral sound accentuated. No mitral diastolic murmurs were heard. The systolic murmur was not transmitted to the left axilla or the back, nor were aortic or tricuspid murmurs heard. Liver was palpable four fingerbreaths below the right costal margin and was non-tender. There was no ascites nor peripheral edema.

The routine hematological, serological, and chemical studies were normal except that the total bilirubin was 1.1 mgm. per cent with less than 0.8 mgm. per cent direct. The electrocardiogram revealed normal sinus rhythm, right axis deviation, a tall R in V1 and depressed ST segments in standard leads 2 and 3, aVf and in V1, V2, and V3. T-3 was inverted. as were the T-waves in V1, V2, and V3. These findings were interpreted as indicative of right ventricular hypertrophy.

Chest fluoroscopy with barium swallow revealed normal peripheral lung fields with no evidence of increased vascularity. Marked right ventricular enlargement was present, and the main pulmonary artery was severely dilated. Prominent pulsations were noted in the hilar regions, but no other specific

PRIMARY PULMONARY HYPERTENSION

Katherine H. Borkovich, M.D.

EVIDENCE THAT pulmonary arterial hypertension exists without apparent cause has been provided by right heart catheterization. Before the introduction of cardiac catheterization, the diagnosis of primary pulmonary hypertension, with few exceptions, was made only at necropsy; and primary pulmonary hypertension had been considered a rare disease. Some authorities, however, feel that essential or primary pulmonary hypertension does not exist except as a result of structural changes in the finer lung vessels. Demonstration by cardiac catheterization of elevated pulmonary artery pressure and normal pulmonary "wedge" or capillary pressure is necessary together with the demonstration of the absence of any intracardiac shunt.

Pathologists had referred to this condition as primary pulmonary arteriosclerosis, right ventricular hypertrophy of unknown origin, primary pulmonary vascular sclerosis, obliterative pulmonary arteriolosclerosis, Ayerza's disease, idiopathic pulmonary hypertension, and pulmonary Raynaud's disease.

E. Romberg, in 1891, described hypertrophy of the right ventricle without demonstrable cause. In 1935, Brenner reported that only 16 cases could be accepted as having primary pulmonary vascular sclerosis. Six years later, Brill and Krygier

chamber enlargement was evident. These x-ray findings suggested primary pulmonary hypertension.

Cardiac catheterization was performed on February 7, 1957, with no evidence of a left-to-right shunt. Pressures in the right ventricle were 108/4/16 millimeters of mercury; in the pulmonary artery, 106/40 millimeters of mercury with a high pulmonary resistance. The capillary pressure was six millimeters of mercury. A femoral artery pressure was 112/70. After the cardiac catheterization, the patient was returned to the ward without incident. Soon afterward, however, she became apprehensive and pale and predicted that one of her "passing out spells" was going to occur. She became pulseless; no cardiac sounds were audible on auscultation. The pupils were widely dilated bilaterally, and bilateral Babinski reflexes were present. The patient's chest was thumped and she was shaken. Within a minute or two, cardiac activity was resumed with a rapid irregular rhythm which, within a few minutes, decreased to 80 per minute. During her unconsciousness, the patient was given oxygen by face mask. The electrocardiogram was obtained about five minutes after the onset of the syncopal episode, while oxygen was being given. The ST depression which had been noted in the first record in standard leads 2 and 3, aVf and V1, V2, and V3 was less marked, and the T-waves were upright and of good amplitude in V2 and V3, whereas they were inverted in the previous record.

During her hospitalization, the patient was treated with maintenance digitalis, 0.1 gm. daily, a 0.5 gm. sodium chloride diet, Thiomerin®, and Demerol®. She was discharged on this same medication. In view of the reported experience of untoward reactions to barbiturates by Doctors Scott R. Inkley, Louis Gillespie, and Robert K. Funkhouser in the **Annals of Internal Medicine**, August, 1955, all barbiturates were withheld. The patient lost 13 pounds on this cardiac regime, and the liver was not palpable at the time of discharge. After the syncopal episode, she was placed on Pro-Banthine®, 15 mg. four times a day, with the hope of at least diminishing some reflex vagal mechanisms which might be contributing to the production of her syncope.

A year previously, Henry Bahnson, M.D., had performed bilateral denervation of the lungs on a 37-year-old white woman with primary pulmonary hypertension and excruciating chest pain in addition to right-sided heart failure. In view of the satisfactory experience obtained, the same surgical procedure was planned for the patient described here.

She was readmitted on April 26, 1957, for re-evaluation and possible denervation of the lungs. Only two more syncopal episodes had occurred; however, she required more Thiomerin® than she did before her first admission. The findings in the physical examination were essentially unchanged; the blood pressure was again 100/80.

Pulmonary denervation was attempted on May 13, 1957. Preoperative medication was 75 mg. of Demerol® and 0.4 mg. of scopolamine. She was given cyclopropane and oxygen and 5 cc. of pentothal. Her blood pressure slowly declined, and her color became poor shortly after the skin incision was made. Cardiac rate became extremely slow, with a sinus bradycardia. The electrocardiogram had been monitored continuously.

Since the cardiac action continued to deteriorate, the patient was rolled over on her back in an attempt to improve her breathing. Initially, stimulation was given by quick jerks on the chest without opening it, because it was felt that opening the chest would produce greater embarrassment than already existed and that any operative procedure or whatever else could be done at that time would not give immediate benefit. Ventricular fibrillation occurred, whereupon the chest was immediately opened, and the heart was found to be severely dilated with poor tone. The patient failed to respond to intracardiac epinephrine, calcium chloride, Isuprel®, cardiac massage, and electrical defibrillation.

Autopsy examination revealed pulmonary arteriosclerosis and arteriolosclerosis. There was dilatation of the intrapulmonary arteries with dilatation and pronounced right ventricular hypertrophy as well as dilatation and moderate hypertrophy of the left ventricle. Multifocal scars appeared in the myocardium of the left ventricle. The liver and pancreas showed slight chronic passive congestion, and peculiar focal scarring was found in the kidneys with foci of tubular regeneration. The cerebral cortex had areas of atrophy and focal encephalomalacia.

Clues in its diagnosis
Conjectures on its cause
Recommendations for its treatment

analyzed the 20 cases that were available from the literature, and in 1952, Parmley and Jones reviewed the findings in the 31 cases of primary pulmonary arteriosclerosis reported up to 1950. Since 1950, more than 60 cases in adults and more than 20 in children have been reported.

The diagnosis of primary pulmonary hypertension must be suspected in any patient who develops progressive exertional dyspnea and weakness, syncope on exertion, left chest pain, and who has evidence of right ventricular hypertrophy, pulmonary arterial dilatation, decreased vascularity of the peripheral lung fields, combined with high right ventricular and pulmonary arterial pressures and normal pulmonary capillary pressure in the absence of pulmonary disease. Primary pulmonary hypertension is usually seen in patients between 20 and 40 years of age, although it occurs in children and infants as well. The ratio of females to males over the age of 12 is four to one among the reported cases, but among children under 12, the distribution between the sexes is about equal.

Physiologic studies have revealed normal pulmonary function and moderate to severe pulmonary hypertension with the pulmonary artery mean pressure exceeding 40 mm. Hg. in all but a few reported cases. The cardiac index was usually subnormal. Total pulmonary and pulmonary vascular resistances were greatly increased. The pul-

monary capillary pressure was normal. Therefore, the increased resistance must occur between the pulmonary artery and pulmonary capillaries because of organic and, probably, functional narrowing of small pulmonary arteries and arterioles.

The most constant symptom in the reported cases was exertional dyspnea, which was apparently caused neither by pulmonary congestion nor by impairment of pulmonary function. It has been postulated that pulmonary hypertension per se might cause dyspnea by reflex stimulation of the respiratory center. Increased pulmonary vascular resistance with reduction of the pulmonary vascular bed may be another contributing factor. Syncopal episodes were present in more than half of the cases reported and were usually precipitated by exertion. The mechanism of the syncope is not clear, although it may be due to decreased cerebral blood flow secondary to decreased cardiac output or to vasovagal reflex. Dressler suggested that the syncope is caused by a reflex mechanism originating in neuroreceptors in the wall of the pulmonary artery, using the vagus nerve as an afferent pathway, and resulting in a fall of the systemic blood pressure and inhibition of the heart beat.

Chest pain, frequently simulating angina pectoris, was present in almost half of the reported cases. This pain may be directly attributed to severe pulmonary hypertension associated with dilatation and stretching of the pulmonary arteries; it may, on the other hand, be due to right ventricular myocardial ischemia. Since coronary blood flow is greatest during diastole, it is possible that the marked rise observed in right ventricular end diastolic pressure might impede the coronary circulation, which could account for the symptoms of angina and could precipitate ventricular fibrillation or cardiac standstill with resultant syncope. Autopsy studies reported in 1954 by Doctors Herbert Gold, Eliot Corday, and Leo Kaplan suggested that the dilated pulmonary artery and its main branches may compress the coronary vessels, which are in such close proximity, and that this might actually interfere wtih the coronary flow. D. T. Dresdale, M.D., reported on a patient who experienced excruciating substernal pain while performing a standard step-up exercise test and 30 seconds later became unconscious and finally cyanotic. On another occasion, this same patient developed chest pain while performing an exercise tolerance test, and the electrocardiogram revealed depression of the ST segments. Brenner reported on an 11-year-old boy who died during an attack of chest pain, and Killingsworth reported on a 10-year-old boy who died after an episode of severe epigastric pain which radiated to the arms.

In neither of these cases nor in the majority of the other reported cases of primary pulmonary hypertension has coronary sclerosis been demonstrated. It is of interest, too, that angina and syncope do not occur in secondary pulmonary hypertension, although increased pulmonary artery pressure and right ventricular hypertrophy are present in both primary and secondary pulmonary hypertension. The effect of Priscoline,® a sympatholytic and adrenolytic agent, in reducing the elevated pulmonary artery pressure with a concomitant increase of blood flow in primary pulmonary hypertension suggests that an overactive autonomic nervous system may contribute to this disorder. Harris, in 1957, also reported significant reduction of the pulmonary artery pressure following acetylcholine injection in one patient. These observations would suggest that although organic changes may be present, functional vasoconstriction may be a contributing factor, especially early in the disease.

Pathologic findings revealed hypertrophied and dilated right ventricle, enlarged right atrium, and normal left side of the heart. A patent foramen ovale was present in a small number of cases. The main pulmonary arteries were enlarged, and atheromatous changes were observed in most cases.

Histologically, subendothelial or intimal proliferation of small pulmonary arteries and arterioles was present in all but a few patients. Hypertrophy of the media was described in about 80 per cent of the reported cases; in all, moderate to severe reduction in the lumina of the small vessels had occurred. Necrotizing angiitis was described in several cases. The nature of the thrombo-embolic changes in the lumina of the small pulmonary arteries is not clear. It is well known that repeated episodes of pulmonary thrombosis and embolism may lead to considerable increase in pulmonary vascular resistance and pulmonary hypertension. It is also possible, though, that long-standing pulmonary hypertension with weakening of the vessel wall may predispose to thrombo-

embolism of the pulmonary vessels. Lewis Dexter, M.D., in 1957, described a patient who had been incapacitated by recurrent pulmonary emboli which had continued even after ligation of the vena cava. The patient was given anticoagulants for only two months. This treatment apparently stopped the thrombo-embolic process, for the patient improved and remained fairly active, although not normal, for seven years until right ventricular failure ensued.

Long-term anticoagulant therapy has been recommended by some physicians as the only form of therapy that could possibly help such patients. Oral administration of Priscoline® has not been helpful. Sympathectomy, performed on a limited number of patients, has also been disappointing. I, personally, had one patient who, by sympathectomy, was symptomatically relieved of her intractable chest pain. She was able to go through an uneventful pregnancy one year after denervation, but she died suddenly a year after delivery. Inhalation of 100 per cent oxygen over a prolonged period has also been unsuccessful in all but one reported case. Steroid therapy has been tried in a limited number of patients with little success. Recently Isuprel® has been shown to lower elevated pulmonary artery pressure; therefore, sublingual Isuprel® therapy might be tried in such patients.

The pathogenesis of primary pulmonary hypertension is still unknown. It may be due to persistence of the fetal anatomy of the pulmonary vessels, excessive pulmonary vasomotor tone, or other unknown factors. Neuro-hormonal imbalance may produce increased pulmonary vasoconstriction, or repeated thrombo-embolic episodes may occur, causing obstructive lesions in the pulmonary arteries. The possible role of serotonin in this disease has recently been considered.

Howarth and Lowe showed that exercise can increase the pulmonary vascular resistance and, thereby, further reduce the already critically low cardiac output with a resultant fall in systemic blood pressure, syncope, and, in some cases, sudden death. Hypoxia has been demonstrated in the pulmonary artery just before a syncopal attack. This may be one factor causing the increase in pulmonary vascular resistance. Sudden deaths have occurred during or shortly after cardiac catheterization and induction of anesthesia and following simple vena puncture. Two deaths were reported in association with the administration of barbiturates. Sven Rune Johnson, in 1951, reported a striking drop in cardiac output and a slight increase in peripheral resistance with barbiturate anesthesia. P. Winchel also reported a decrease in cardiac output and an increase in peripheral resistance after the administration of sodium amytal in normal patients. It has been recommended, therefore, that barbiturate or general anesthesia be avoided in such patients in favor of local or spinal anesthesia. In view of the foregoing factors, any investigatory or surgical procedure may be hazardous in such a patient.

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SERUM LACTIC DEHYDROGENASE

in the Diagnosis of Acute Myocardial Damage

CORONARY ATHEROSCLEROSIS is a disease with a wide spectrum of activity. At one end of the spectrum is the patient who develops angina only with severe exertion; at the other end is the patient who develops a frank transmural myocardial infarction at rest. In between are many intermediate stages. Various terms have been used to designate these intermediate stages of coronary atherosclerosis. Such terms as coronary insufficiency, coronary failure, intermediate coronary syndrome, subendocardial infarction, intramural infarction, mild infarction, and "non-specific acute T wave inversion, suspect acute myocardial injury" have all found favor with some. The electrocardiogram in the intermediate stage of coronary atherosclerosis may be within normal limits or may show ST T changes with varying degree of specificity.

Angina of effort usually can be satisfactorily diagnosed by a careful history with objective support from a Master's tolerance test when necessary. Likewise, frank transmural infarction usually can be satisfactorily diagnosed from the clinical syndrome and serial electrocardiograms. In contrast, diagnosis of the intermediate stages of coronary atherosclerosis is often difficult. When it is possible to make a diagnosis with reasonable certainty, it is often difficult to assay the extent of the acute myocardial damage which has occurred and to determine the proper therapeutic regime which should be recommended to the patient.

The need for auxiliary diagnostic aids in the diagnosis of acute myocardial damage has been obvious. In response to this need, a series of laboratory procedures has been proposed, in recent years, as diagnostic aids. Procedures which have been investigated include studies of plasma fibrinogen concentrations; determinations of various enzymes, such as serum glutamic oxaloacetic transaminase, serum glutamic pyruvate transaminase, lactic and malic acid dehydrogenase, and serum aldolase; the C-reactive proteins; serum muco pro-

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An evaluation of the colorimetric determination of serum lactic dehydrogenase as an aid in the diagnosis of acute myocardial damage

teins, serum polysaccharides, and others (1). Of these, the serum glutamic oxaloacetic transaminase (SGOT) and the serum lactic dehydrogenase (SLD) have gained widespread popularity, especially the former (2).

The SGOT and SLD determinations share many attributes. Both GOT and LD are intracellular enzymes widely distributed in normal human tissues and body fluids. Both enzymes are heavily concentrated in heart muscle. The activity of GOT is 5,000 to 10,000 times greater, and that of LD is 3,000 times greater in the myocardium than in the serum. Myocardial necrosis results in the release of both enzymes from the injured myocardial cells and concentration in the serum. Following myocardial necrosis, peak activity of both enzymes occurs in 12 to 48 hours and falls to or toward normal in two to five days. The height and duration of activity of each bears a semi-quantitative relationship to the extent of the myocardial necrosis. Both tests have been shown to have a high degree of accuracy in acute myocardial necrosis. In more than 2,000 cases of infarction reported in the literature, the SGOT has been positive in 96 per cent (3). In more than 200 cases, the SLD has been positive in 89 per cent (3). Neither test has been positive in uncomplicated angina; nor is either a specific test for myocardial damage, since each may be elevated in disease processes involving organs other than the heart. In common, the SGOT and SLD may be elevated in cerebral thromboembolic disease with infarction, in acute hepatitis, in hemolytic states, in leukemias and malignant lymphomas, and in skeletal muscle injury, whether from surgical or accidental trauma or from diseases of muscle such as dermatomyositis or progressive muscular dystrophy (4, 5, 6, 7, 8). In general, in these conditions, the curve resulting

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from serial determinations will be distinct from that seen in myocardial necrosis; but this is not invariably so.

There are notable differences in the characteristics of GOT and LD. GOT is considered to be intimately connected to the cell nucleus and LD to the cell cytoplasm, resulting in a generally increased sensitivity of the LD determination as compared with the GOT (9). In contrast to the SGOT determination, the SLD determination is easily affected by hemolysis (10). Poor technique in blood collection, therefore, may lead to falsely positive SLD determinations. The SGOT may be elevated in chronic liver disease, such as active Laennec's cirrhosis, in pancreatic necrosis, and in obstructive jaundice, whereas the SLD is normal or minimally elevated in these conditions. On the other hand, the SLD is more markedly and more consistently elevated in acute leukemia, in chronic granulocytic leukemia, and in disseminated carcinoma and lymphomata (6, 8). In acute myocardial necrosis, important differences in SGOT and SLD activity have been reported. The SGOT activity increases proportionately more than the SLD activity (10, 11); however, the SLD activity has generally been found to persist two or three days longer (10).

We will report here the results of an attempt to evaluate the usefulness of the colorimetric SLD determination as compared with the more popular SGOT in the diagnosis of acute myocardial damage. The published reports on the subject of serum lactic dehydrogenase in acute myocardial infarction are based almost exclusively on a spectrophotometric procedure for determination of the serum lactic dehydrogenase. Since the spectrophotometric procedure is not available to the average small clinical laboratory, there is a need for an evaluation of the colorimetric determination which has been recommended for use in the small hospital without a spectrophotometer.

MATERIALS AND METHODS

ONE HUNDRED AND NINE consecutive patients admitted to the Franklin Square Hospital during 1958-59 with a diagnosis of acute myocardial infarction or suspected myocardial infarction were studied. On each patient the SGOT, the SLD, the C-reactive protein, the leukocyte count, and the sedimentation rate were determined on admission and daily until either the SGOT or the SLD became positive or until both tests remained consistently negative for a period of days. When either the SGOT or the SLD became distinctly positive, determinations were carried out on each alternate day until values returned to the normal range. Temperatures were taken four times daily so that an adequate temperature curve would be available for each patient. Serial EKG's were done on each patient at varying time intervals as indicated by the characteristics of the individual patient. A careful history and physical examination were carried out on each patient.

The SLD and SGOT determinations were performed in the laboratory of the Franklin Square Hospital. The SLD was determined by the colorimetric procedure developed in the laboratories of the Sigma Chemical Company and described by Berger and Broida (13). The SGOT was determined by the colorimetric procedure described by Riteman and Frankel (15). For the SGOT determination, a value in excess of 40 units was considered abnormal; and for the SLD determination, a value over 680 units. In this study both the SLD and the SGOT were measured colorimetrically, which method does not involve the use of an expensive U.V. spectrophotometer and, therefore, may be utilized by the small clinical laboratory. The developers of this procedure suggest that the units obtained by the spectrophotometric method of Wroblewski (5) may be interchanged with the units obtained by the colorimetric procedure (13).

The patients were divided into groups as follows. Group I consisted of those patients with unequivocal evidence of infarction. Inclusion in this group required that the patient have the clinical syndrome of infarction and characteristic EKG changes in serial tracings, including abnormal Q waves. Group II consisted of those patients who probably had an infarction. These patients had a clinical syndrome suggestive of an infarction and evolving ST T abnormalities in serial EKG's compatible with acute myocardial injury, although abnormal Q waves were not present. Only patients with distinctly characteristic ST T changes were included; those with minor changes or with changes which could be explained on some other basis were excluded. Group III consisted of patients in whom, after

TABLE I

	Group I Definite Infarction	Group II Probable Infarction	Group III Indefinite	Group IV No Infarction
NUMBER OF CASES	55	8	22	15
SGOT POSITIVE / SLD POSITIVE	32 (58%)	2	5	0
SGOT POSITIVE / SLD NEGATIVE	0 (0%)	0	1	0
SGOT NEGATIVE / SLD POSITIVE	12 (22%)	4	5	0
SGOT NEGATIVE / SLD NEGATIVE	10 (18%)	2	12	15

Table I compares the results of the SGOT and SLD determination in the four groups of patients: Group I—unequivocal infarction; Group II—probable infarction; Group III—indefinite i.e. it could not be definitely stated that patient did or did not have myocardial necrosis; Group IV—no infarction i.e. after study, it was clear that there was no reason for further suspicion that myocardial necrosis had occurred.

study, there was doubt as to whether infarction had occurred. Group IV consisted of patients who, on the basis of clinical findings and serial EKG tracings, could be confidently classified as not having had an infarction. In all groups, care was taken to rule out other causes for the patients' symptoms, such as hiatus hernia, cholecystitis, or pancreatitis. Care was also taken to rule out other causes for an elevation in temperature, leukocyte count, SGOT, and SLD.

RESULTS

TABLE I SHOWS the results of the SGOT and SLD determinations in the 109 cases. Of the 109 patients, 55 were classified as definitely having had a myocardial infarction, eight as probably having had an infarction, 22 as being in the indefinite group, and 15 as not having had an infarction. Of those with a definite infarction, 32 had positive SGOT and SLD determinations, and ten had a negative SGOT and SLD. Twelve had a positive SLD determination, although the SGOT was negative. In no instance was the SLD negative when the SGOT was positive. Comparing the SGOT determination directly with the SLD, the SGOT was positive in 58 per cent of 55 cases of definite infarction and the SLD in 80 per cent. Hence, the SLD appeared to be significantly more sensitive in this group of patients. This study yielded no false positives, although it has been considered a liability of the SLD determination that faulty blood collection techniques with hemolysis of red blood cells may give falsely elevated results for the SLD determination.

Table II shows the duration of activity of abnormal SGOT determinations as compared with SLD determinations. Whereas the SGOT determination remained elevated to an abnormal level for five days or longer in only 14 per cent of cases, the SLD remained elevated five days or more in 48 per cent of cases.

DISCUSSION

ALTHOUGH THE NUMBER of cases in this study is not great, we feel that the results are valid for the following reasons. In contrast to the study which is done retrospectively by an investigator who studies charts "pulled" from the files on patients whom he himself did not see, a method of investigation was agreed upon in this study, and the patients were studied as they entered the hospital. Consecutive admissions for a suspected infarction were studied. All patients

TABLE II

Number of Days of Abnormal Elevation of Enzyme	Cases SGOT	Cases SLD
1	30	12
3	8	11
5	3	7
7	3	9
9	0	4
11	0	1

Table II shows duration of abnormal elevation of SGOT and SLD in patients with acute myocardial infarction. Forty-four cases are included in this series. Serial determinations of both serum enzymes were done every other day until the values returned to normal level.

were observed clinically by one of the authors, and all EKG's were reviewed by each author.

The incidence of positive SGOT and SLD determinations is less than that generally reported, particularly with respect to the SGOT. This fact may be the result of a decreased sensitivity of the colorimetric procedure for determining enzyme activity as compared to the spectrometric method. We believe, however, that, to some extent, the lower frequency of positive reactions is a reflection of the delayed admission of many patients to the hospital. Such delays are often necessitated by a shortage of hospital beds; in some instances, delay is the result of the patient's failure to seek early medical attention, and in other cases, the delay occurs because the physician or the patient elects to attempt treatment at home.

Although in this study the reliability of the SLD determination was 12 per cent greater than the SGOT determination, the conclusion that the SLD determination is more sensitive is not justifiable, since our statistics do not include exact information on the time relationship between the occurrence of infarction and the first determination of enzyme activity. It is our impression, that the apparent increased sensitivity of the SLD determination is a reflection of the fact that elevation of the SLD persists three to four days longer than do elevations in the SGOT.

SUMMARY AND CONCLUSIONS

1. One hundred and nine consecutive hospital admissions with actual or suspected myocardial infarction were studied with particular reference to the colorimetric SGOT and SLD determinations as aids in diagnosis.

2. In 55 cases of unequivocal infarction, both the SGOT and SLD determinations were abnormally elevated in 32 (58 per cent).

3. In an additional 12 cases (22 per cent), the SLD was abnormally elevated when the SGOT was in the normal range. Thus, the SLD determination was positive in 80 per cent of cases as compared with 58 per cent for the SGOT.

4. In no case was the SLD determination negative when the SGOT was positive.

5. There were no false positives for either determination.

6. Whereas the SGOT determination remained abnormally elevated for five days or longer in only 14 per cent of cases, the SLD remained abnormally elevated for five days or longer in 48 per cent of cases.

7. The SLD determination is a useful aid in the diagnosis of acute myocardial necrosis. It appears to have an important advantage over the SGOT determination in that it remains abnormally elevated for three to four days longer, thereby giving rise to a higher percentage of accuracy in patients in whom, for one reason or another, the attempt to confirm the diagnosis of myocardial necrosis by a determination of enzyme activity is delayed from four to seven days after infarction occurs.

8. Our figure of 80 per cent positive SLD determinations in 55 cases of unequivocal myocardial infarction is lower than that generally reported in the literature and considerably lower than the 100 per cent correlation reported recently by Snodgrass et al (16). This fact suggests that the colorimetric determination of SLD as done in a typical small clinical laboratory is less sensitive than the spectrophotometric procedure.

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Important Dates

APRIL 26, 27, and 28, 1961

ANNUAL MEETING

of

MEDICAL and CHIRURGICAL FACULTY

The Alcazar, Cathedral and Madison Streets, Baltimore

The 163rd Annual Meeting of the Medical and Chirurgical Faculty of the State of Maryland will be held at the Alcazar in Baltimore on Wednesday, Thursday, and Friday, April 26, 27, and 28, 1961. The Committee on Scientific Work and Arrangements, under the chairmanship of William E. Grose, M.D., is arranging another outstanding and educational program with many speakers of national eminence; to mention a few, Merrill A. Bender, M.D., of the Roswell Park Memorial Institute in Buffalo; Perry S. MacNeal, M.D., of Philadelphia; William S. Jordan, Jr., M.D., of the University of Virginia School of Medicine; Harry M. Rose, M.D., of Columbia University; Russell B. Roth, M.D., of Erie; Robert H. Parrott, M.D., of the Children's Hospital of the District of Columbia; Allan C. Barnes, M.D., of The Johns Hopkins University School of Medicine; and William S. Stone, M.D., of the University of Maryland School of Medicine. A feature which has proven popular in the past will be held again this year on Wednesday evening—a medicolegal symposium.

There will be many technical exhibits and several scientific exhibits which, it is hoped, our members and guests will plan to visit, as the time spent for this will be well repaid by the information gained.

The following is an outline of the schedule for the Annual Meeting:

- *Business sessions*—Wednesday morning, April 26, and Friday afternoon, April 28. All resolutions to be presented to the House of Delegates must be in the Faculty office by March 1.
- *Scientific Sessions*—Wednesday afternoon and evening, April 26; all day Thursday, April 27; and Friday morning, April 28.
- *Round Table Luncheon*—Thursday, April 27, Park Plaza Hotel.
- *Presidential Dinner and Meeting*—Thursday, April 27, Sheraton Belvedere Hotel.
- *Woman's Auxiliary Meeting and Luncheon*—Wednesday, April 26, Sheraton Belvedere Hotel.

MARK THESE DATES ON YOUR CALENDAR AND PLAN TO ATTEND

SCIENTIFIC SESSIONS—BUSINESS MEETINGS—EXHIBITS

WEDNESDAY, APRIL 26

THURSDAY, APRIL 27

FRIDAY, APRIL 28, 1961

COMMITTEE FOR THE

STUDY OF PELVIC CANCER

(Under the auspices of the Medical and Chirurgical Faculty and the Maryland Division of the American Cancer Society)

Howard W. Jones, Jr., M.D.
Chairman

The Committee for the Study of Pelvic Cancer met with the general staff at Provident Hospital on October 20, 1960. On November 16, a meeting was held with the Anne Arundel County Medical Society.

Abstract of case discussions:

The patient was a 48-year-old widow, gravida 4-0-0-4. She gave a history of regular periods until December 1959, when she had prolonged and profuse bleeding. She consulted her family doctor at this time. A pelvic examination was made, and she was advised to go to the hospital for further examination. Her physician prescribed medication which stopped the bleeding, and the patient deferred going to the clinic. Her periods from December to March were at regular intervals but were profuse. She again consulted her doctor and was again advised to go to the hospital. Beginning in March, she had almost daily bleeding, which varied in amount. On April 30, she returned to her doctor and again was given a note of referral to the hospital. She went to the hospital on May 4 and was admitted the following day.

Diagnosis: Carcinoma of the cervix, international classification, stage II.

Treatment: Radiation therapy.

PHYSICIAN: At the time the patient first went to the doctor, why did he give medication if he advised the patient to go to the hospital? Even though he did not delay, he contributed to the patient's delay by giving her medicine which relieved her symptoms; thus she did not go to the hospital.

CHAIRMAN: I think that is a good point. It would be helpful to know what the medication was. It might have been iron to relieve anemia or something of that kind. I would feel that the fact that the bleeding stopped was coincidental and not related to the medicine. At least, I do not know of any medicine that stops bleeding from carcinoma.

PHYSICIAN: We know that the patient was advised to go to the hospital, but do we know what the physician had in mind; what his examination revealed? Did he suspect malignancy?

CHAIRMAN: We unfortunately do not have any further information from the doctor. We attempt to get this information, but the doctors do not always reply to the letters.

PHYSICIAN: I think we need to add more to these histories and to know what the examinations reveal.

COMMITTEE MEMBER: In this case, the physician apparently did not delay at all in advising the patient to go to the hospital. It is not specifically mentioned on the abstract we have today, but I understand that the patient was given a note of referral to the hospital at the time of each visit to the doctor. I am sure we have all treated patients in the same way—found that they needed treatment but patted them on the shoulder and said, "Don't you worry; you go to the hospital and you will be taken care of." I think this physician is entirely blameless. He examined the

patient and advised that she go to the hospital but she did not follow this advice. I think this is entirely patient delay.

COMMITTEE MEMBER: There was a lapse of time from December to May before the diagnosis was made. During this time the patient returned to the physician several times. Did he at any time take a smear or a biopsy?

CHAIRMAN: His note of referral to the hospital does not indicate that he did.

PHYSICIAN: If you see a patient with a suspected malignancy, obviously you cannot always say to the patient, "I think you have cancer;" but tell her husband, tell her family, tell her sister or her brother—tell somebody who can help in getting her to follow advice and go to the hospital.

CHAIRMAN: There are, undoubtedly, ways and ways of getting a patient to cooperate. Without alarming the patient, I think you can be emphatic that she needs further examination or treatment and make arrangements for her to be seen at a hospital clinic, go to a specialist, or do whatever is indicated.

PHYSICIAN: I think a physician has to assume responsibility for doing all that he can to see that the patient goes to a hospital or has further examination as seems necessary.

PHYSICIAN: I said before and did not get much agreement, and I say again that I do not think medication should be given if a patient is to be referred to another physician or sent to the hospital. I am a general practitioner, and when I see a patient I ask myself, "Is this something I can handle or not?" If I think it is something I cannot handle, I refer the patient to another doctor or to the hospital; and I do not give medicine if I am referring the patient. If the symptoms are relieved, the patient may rely on the medicine and not go to the hospital or to another doctor. Also, medication may mask the symptoms and make the diagnosis more difficult.

CHAIRMAN: Shall we vote on this case as to patient delay or patient and physician delay?

FINAL VOTE: Patient delay.

The patient was 65 years of age, married, gravida 3-0-1-3. She had an uneventful menopause in 1940 and no post-menopausal bleeding. About March 1960, she began to notice intermittent pain in the low back, radiating down legs, and pain in the lower abdomen. She consulted a physician soon after the onset of these symptoms and was told that she had arthritis. She was later referred for x-rays of her back and was again told that she had arthritis. The pain became increasingly severe, and the patient returned to her physician many times. She was treated with various medicines with no improvement. In mid-July, she went to a hospital clinic regarding the pain. Following a pelvic examination, the patient had very profuse bleeding, having had had no bleeding previous to this time. She was immediately referred to a second hospital for diagnosis and treatment.

Diagnosis: Carcinoma of the cervix, international classification, stage III a.

Treatment: Cobalt therapy and radium application.

CHAIRMAN: Would anyone care to comment on this case? It is rather unusual in that apparently there were no symptoms except back pain, pain in the lower abdomen and in the legs.

PHYSICIAN: Do you believe her story?

CHAIRMAN: She was very firm about it, and the impression was that the history was reliable.

COMMITTEE MEMBER: I think this is one of the saddest things we have to deal with. A patient has no symptoms referrable to the pelvis and is later found to have advanced pelvic carcinoma.

COMMITTEE MEMBER: I do think this patient should have had a pelvic examination before she did. Even though her only symptom was back pain, this was apparently fairly severe and increasing. In cervical carcinoma there is usually abnormal bleeding of some kind, often postcoital

bleeding and bleeding after douching; but this is not always true. I recently had a postmenopausal patient who had had one day of slight bleeding—no other symptoms. A dilatation and curettage was done, and she was found to have a carcinoma of the endometrium which proved to be extensive.

PHYSICIAN: Do we have any other information as to the examination of this patient. Was there gross carcinoma?

CHAIRMAN: The hospital record describes the cervix as showing an exophytic growth infiltrating the left fornix. The parametria was said to be infiltrated to the pelvic wall on the left and less on the right.

PHYSICIAN: I think this patient should have had a medical examination. She certainly needed some further investigation of her symptoms even though they did not definitely indicate a gynecological examination. Probably she did have arthritis, but most 65-year-old women do.

COMMITTEE MEMBER: I don't think that any physician in general practice can be indicted for not doing a pelvic. The only complaint was backache.

COMMITTEE MEMBER: She apparently had a definite pattern of pain: in the back and radiating to the lower abdomen and down the legs. This persisted and increased over some length of time. I think she should have had further investigations.

COMMITTEE MEMBER: This patient did not go to her doctor only once with this complaint; she returned to him many times with apparently increasingly severe symptoms.

COMMITTEE MEMBER: Of course, I think every woman patient should have a pelvic examination when she goes to a doctor for examination whether or not she has symptoms referrable to the pelvis. If you are going to pick up the disease early, the only way to do it is by pelvic examination. This case strongly emphasizes that fact.

Summary

Total cases to November 1, 1960	**2133**
Classification:	
No delay	**752**
Asymptomatic detected cases	**69**
Patient delay	**867**
Physician delay	**153**
Patient and physician delay	**143**
Institutional delay	**49**
Institutional and physician delay	**11**
Institutional and patient delay	**47**
Institutional, patient and physician delay	**4**
Delay due to laboratory error	**7**
Inadequate or improper treatment	**21**
Unclassified to date	**10**

Newly Licensed Physicians

At a reciprocity meeting on November 4, 1960, the Board of Medical Examiners licensed the following physicians to practice medicine and surgery in Maryland:

Bridges, Berly Elliot, Jr., Texas
Feinberg, Richard Justin, Illinois
Feringa, Earl Robert, Michigan
Lammers, Ann Hyacinth, Nebraska
Masi, Alfonse Thomas, National Board
McMahon, Edmund Brown, Vermont
Odom, Emwood Earl, New York

ALLEGANY-GARRETT COUNTY MEDICAL SOCIETY

LESLIE E. DAUGHERTY, M.D.

Journal Representative

ALLEGANY COUNTY LEADS IN MEDICAL CARE COSTS

Health services in Allegany County cost $848,849 during the 1959-60 fiscal year. This sum tops medical care costs in all other Maryland counties, regardless of size and population. Of the budget, $760,836 came from state and federal funds and $88,013 (10 per cent) came from Allegany County funds.

The Allegany County funds included $83,413 spent for general local health services, $4,000 for chronic disease hospital care, and $600 for outpatient services in local hospitals.

Medical care costs surpassed by $194,553 the previous year's total of $157,681.46.

CARLTON BRINSFIELD HEADS MEMORIAL HOSPITAL MEDICAL STAFF

Carlton Brinsfield, a prominent young surgeon of Cumberland, was elected president of the Memorial Hospital medical staff for 1961. A graduate of the University of Maryland School of Medicine, he served his surgical residency at Maryland General Hospital, Baltimore. He has been located in Cumberland since 1954 and is the immediate past vice-president of the Allegany-Garrett County Medical Society.

Dr. Brinsfield is married to the former June Hopkins, of Belair, Maryland. They have three children.

PERSONALS

Leo H. Ley, Jr., M.D., Cumberland, attended the Interstate Postgraduate Medical Convention, held in Pittsburgh October 31 through November 4.

Clay E. Durrett, M.D., has been elected to the board of directors of the Cumberland Savings Bank. A graduate of the University of Maryland Medical School, Dr. Durrett started his practice in Cumberland in 1931.

Leslie E. Daugherty, M.D., moderated a round-table discussion on "Amateur Photography in the Tri-State Area," held recently at Frostburg State Teacher's College.

At a meeting of the Cumberland Gephart School Parent-Teacher's Association, **Gina Glick, M.D.,** spoke on the newly-established poison center at Sacred Heart Hospital. This talk was sponsored by the Woman's Auxiliary to the Allegany-Garrett County Medical Society.

ANNE ARUNDEL COUNTY MEDICAL SOCIETY

SAMUEL BORSSUCK, M.D.

Journal Representative

The Anne Arundel County Medical Society held its regular meeting September 21, 1960, at Carvel Hall, Annapolis. After cocktails and dinner, Mr. Farley, of Merrill, Pierce, Fenner and Smith, told the group "What the Doctor Should Know About Stocks and Bonds."

Doctors George Settle, Harvey Butt, James Hayes, Ernest Leipold, Albert Cooper, and **Joseph Cooper** were introduced to the Society as new and transfer members from the Baltimore City Medical Society.

As part of the business meeting, the delegates to the Semiannual Meeting of the Medical Faculty gave their report. A lengthy and interesting account of the discussion at the Ocean City meeting concerning Blue Cross and Blue Shield was made by Delegates **Lyons** and **Hawkins.** After some discussion the motion was made and passed by the Anne Arundel Medical Society recommending that:

1—There be no further expansion of professional services under Blue Cross.

2—Any expansion of professional service be under Blue Shield.

3—There be separate directors for Blue Cross and Blue Shield.

4—The medical profession not allow itself to be put in the position of policing in connection with insurance programs.

5—The Blue Cross policy be rewritten in such a manner that police action on the part of the medical profession would not be required.

The president called attention to the emergency medical service which had been organized in the county, which to date had been functioning satisfactorily.

A committee, appointed by the president, is to investigate the possibility of putting into action a plan for mutual coverage of members who become disabled for a period of time.

Dr. Yun, who is now practicing in Severna Park, was elected to membership in the County Medical Society.

The November meeting of the Anne Arundel County Medical Society was held in the Galaxy Room at the Friendship Airport on November 16, 1960. The after dinner program was conducted by the Medical and Chirurgical Faculty's Committee for the Study of Pelvic Cancer.

Doctors Sina, O'Herlihy and **Scheye** were elected to the County Medical Society, **Doctors O'Herlihy** and **Scheye** being transfers from Baltimore City Medical Society.

BALTIMORE CITY MEDICAL SOCIETY

CONRAD ACTON, M.D.

Journal Representative

The Executive Board held its regular meeting on Election Day, Tuesday, November 8. **Moses Paulson, M.D.,** chairman of the Constitution and Bylaws Committee, presented the final revision of our Bylaws to conform to those of the Faculty and gave the Board a preview of the model to be presented at the next Society meeting.

The secretary, **Joseph D. B. King, M.D.,** having recovered from his illness, reported on matters of old business. Of particular interest among these was one regarding insurance matters. The Medical Faculty has selected an insurance carrier other than the one which we have heretofore recommended. In its report, accepted at the last meeting, the Com-

mittee on Insurance Matters had recommended that when the Faculty selects its carriers, the City Society should go along. Members of the Board were quick to point out that, in this instance, a change of carriers might cause considerable hardship to some of our members who, although having been accepted by our presently recommended carrier, might have difficulty in getting insurance with the other company. A lively discussion ensued as to how this situation could be met without causing hardship to our borderline and poor risk members.

Mr. G. C. A. Anderson, our legal counsel, in an opinion regarding re-statement of the purposes of the Society as urged by the retiring secretary last year, cogently pointed out ways in which our tax exempt status would be jeopardized by such a maneuver. He advised that for the present no change in the purposes of the Society be expressed in the Constitution.

An offer has been made by a reputable drug house to give financial support to our guest speakers. This manufacturer is making similar offers nationally through its Foundation. In the past, the Executive Board has decreed that, while traveling expenses should be paid, honoraria for speakers at the Society meetings are not in order.

In line with the decision at the November meeting that the City Society should speak out regarding expansion of City Hospitals, the summary of the oft-mentioned Steinle Report was sent to the Executive Board members for study. The Board voted that the Society would best be represented by its Executive Committee, designating the president, vice president, secretary, and treasurer as ambassadors to the Board of Estimates hearings on proposed changes at City Hospitals.

Discussion of a novel proposal that physicians' fees be included within the scope of one of the credit card firms livened the meeting. In some localities, dental charges are so included. That medical charges likewise be settled on credit card terms did not seem generally applicable in the Baltimore milieu.

The format of the ballot sheet for the December election was an appropriate Election Day topic. Listing of nominees in alphabetical order was agreed to be the fairest way to present them. Advance notification by mail will indicate to members which names are on the Nominating Committee's slate and which were nominated from the floor. It was felt that the alphabetical sequence would avoid "party lines."

BALTIMORE COUNTY MEDICAL ASSOCIATION

LOUIS DALMAU, M.D.

Journal Representative

The regular meeting of the Baltimore County Medical Association, Inc. was held November 17 at the Penn Hotel, Towson. **Margaret L. Sherrard, M.D.,** presided.

Prepaid Medical Care

Bernard Sollod, M.D., chairman of the Subcommittee on Medical Economics and Legislation, reported the necessity for both the County and the State medical societies to participate in discussions with representatives of steel workers' unions and other unions which may be interested in various proposals to improve the medical care of the union members and their families. He emphasized the need for the Association to do its best to improve existing conditions and to investigate various forms of prepaid medical care to be offered to the unions. Dr. Sollod suggested that the Baltimore County Medical Association follow the example of the Pennsylvania Medical Society, which has already contributed considerably toward these goals. By unanimous approval, the executive secretary of the Medical and Chirurgical Faculty will be invited to the Association's December meeting to inform our members on these activities at state level. Sufficient copies of an informative pamphlet are to be ordered on prepaid

medical care from the Pennsylvania Medical Society for distribution to the members of this Association.

Use of INH in Control of Tuberculosis

Samuel P. Scalia, M.D., director of the Division of TB Control of the Baltimore County Health Department, gave a concise report on "The Use of INH with Positive Skin Tests." Dr. Scalia urged all county physicians to give serious consideration to tuberculin skin testing in children up to the age of three years and in negative reactors at ten years. The Baltimore County Health Department, in cooperation with the United States Public Health Service, is sponsoring this program. The use of INH in children up to the age of three with positive skin test would prevent dissemination of the TB within a positive reactor and possibly propagation of the disease to others. Teenaged negative reactors who become positive reactors within a year also should have INH prophylactic treatment. The Baltimore County Health Department advocates use of prophylactic INH for a period of one year. This therapy will be available through the Baltimore City Health Department for individuals unable to pay for treatment.

New Members

The following were voted into membership: **Alfred J. Shulman, M.D.**, affiliate; **Ronald R. Berger, M.D.** and **Edgar P. Williamson II, M.D.**, transfers from the Baltimore City Medical Society, and **Stanley Felsenberg, M.D.**, as active members.

Disability Under Social Security

Sidney J. Venable, M.D., medical consultant for the Disability Determinations Program. Federal Old Age and Survivors Insurance Benefits (B.O.A.S.I.), spoke on the disability program. Disability as defined by the Bureau is "the inability to engage in any substantial, gainful activity by reason of any medically determinable condition which can be expected to result in death or to be of long, continued, and indefinite duration." Dr. Venable advised that the disability benefits of the B.O.A.S.I. have recently been extended to persons under 50. A physician filing a claim for an applicant must fill out a Standard Medical Report Form, which the applicant obtains from his local Social Security District Office. The applicant is to pay for his own medical examination when initiating the claim.

The examining physician is not expected to rate the disability of the patient, for this is the Bureau's function. The local district office will help an applicant to establish his claim. The claimant's impairment is evaluated in terms of the aforementioned definition. Determining the degree of impairment or severity of the applicant's condition is the responsibility of the State Agency's consultant physician; the final disability decision rests with the Disability Division of the Bureau.

Practicing physicians are urged to provide sufficient medical information or evidence; that is, complete history, physical examination, laboratory data, x-ray findings and other pertinent clinical information which would aid the evaluating medical consultant and the Bureau's team in arriving at a decision of disability. Claims which are denied may be reviewed or reconsidered at the state level and by the Bureau. Dr. Venable emphasized that a scanty medical report usually makes evaluation of the medical evidence difficult and delays the processing of the claim and the evaluation of the alleged disability. The Bureau would have to secure the medical consultation necessary to develop a case whenever the medical evidence is not complete or clear enough to meet the concepts of disability established by the Bureau. A good medical report expedites proper processing and prompt payment of benefits to the claimant.

An excellent movie on Disability Evaluation, sponsored by the American Medical Association, further illustrated the concepts expressed by the speaker.

FREDERICK COUNTY MEDICAL SOCIETY

L. R. SCHOOLMAN, M.D.

Journal Representative

The October meeting of the society was unusual in time, place, and composition: it was held on a Saturday in the ballroom of the Francis Scott Key Hotel, and it was the second command attendance meeting in two years. Members' wives were invited guests, as were such local celebrities as **Dr. Andrew Truxal,** president of Hood College, and **Mr. William T. Delaplaine,** editor of the *Frederick News-Post,* and their wives. They enjoyed the talk by **Richard Te Linde, M.D.,** professor emeritus of gynecology at The Johns Hopkins Medical School. Dr. Te Linde, speaking on "Medico In The Near East," described his experiences as a member of the medical team which worked in Jordan. Colored photograph slides illustrating his topic added to the interest.

The Society welcomed a new member, **Thomas Michael, M.D.,** an otolaryngologist who has moved from Baltimore. We hope he will thrive on the more leisurely pace.

The November meeting was held at the Francis Scott Key Hotel on the 22nd. The speaker of the evening was **Alfred Shulman, M.D.,** a Baltimore psychiatrist who, during the past two years, has been seeing patients in Frederick two days a week. Dr. Shulman discussed "Practical Use of Psychiatric Facilities in Maryland." The interest aroused in his subject was evidenced by the many questions put to him during the discussion period.

MONTGOMERY COUNTY MEDICAL SOCIETY

CHARLES FARWELL, M.D.

Journal Representative

William J. Peeples, M.D., and **Gaston de Lemos, M.D.,** established a new outpatient clinic for followup of Springfield Hospital psychiatric patients.

Gilbert V. Hartley, M.D., president in 1928 of our County Medical Society, was honored by a biographical sketch and photograph in our *Medical Bulletin.*

Harold H. Mitchell, M.D., wrote "Medical Aid for the Aged—What's Next?" for our *Medical Bulletin.*

A number of our members took part in the medical disaster practice, which added greatly to its success. A mock fire in a regional shopping center supposedly produced many casualties, giving the medical and rescue workers an opportunity to exercise their skills in handling community chaos and catastrophe. While patient review and self-criticism revealed areas where cooperation could be improved between firefighters and medical workers—for instance, by routing fire trucks around instead of through the casualty clearing centers—the overall result was favorable. Monitors who have observed similar disaster drills elsewhere in our nation were quoted as rating our efforts excellent. Each physician who took part in these preparations for a community disaster, which we hope never occurs, deserves honorable mention; particularly worthy of praise for his continued interest and determined persistence is **Merrill M. Cross, M.D.**

Our annual dinner dance was held at the Indian Spring Country Club. A good time was had by all!

The Southeastern Chapter of The Society of Nuclear Medicine will hold its second annual meeting on March 10 and 11, 1961. The meeting will take place in the auditorium of The Academy of Medicine, Atlanta, Georgia.

WICOMICO COUNTY MEDICAL SOCIETY

GLADYS M. ALLEN, M.D.
Journal Representative

At the November meeting of the Wicomico County Medical Society, **John Vasconcellos, M.D.,** was accepted for membership on transfer from the Baltimore County Medical Society. He has been working for the Department of Mental Hygiene in Baltimore for the past four years.

The guest speaker was **Richard Shackelford, M.D.,** associate professor of surgery, The Johns Hopkins Hospital; chief of surgery of the Perry Point Veterans Hospital; and author of the three volume text, "Surgery of the Alimentary Tract." Dr. Shackelford gave an interesting discussion on "Recent Advances and Problems in Gastrointestinal Surgery."

WORCESTER COUNTY MEDICAL SOCIETY

FRANCIS J. TOWNSEND, M.D.
Journal Representative

At the quarterly meeting of the Worcester County Medical Society, held in Snow Hill in October, the following officers were elected for 1961: President, **C. Stanford Hamilton, M.D.,** Pocomoke; Vice president, **Norman E. Sartorius, Jr., M.D.,** Pocomoke; Secretary-Treasurer, **Charles W. Trader, M.D.,** Pocomoke; Delegate, **Robert C. LaMar, M.D.,** Snow Hill; Alternate delegate, **Nathaneal R. Thomas, M.D.,** Ocean City; Censor 1960-63, **Robert C. LaMar, M.D.,** Snow Hill; Diabetes Committee 1960, **Paul Cohen, M.D.,** Snow Hill; Diabetes Committee 1961, **G. S. Hamilton, M.D.,** Pocomoke; Journal representative, **Francis J. Townsend, Jr., M.D.,** Ocean City.

T. HOWARD STRAUB APPOINTED COUNSELOR AT MELWOOD

Melwood Farm, Olney, Maryland, announced the recent appointment of Mr. T. Howard Straub as counselor and program administrator. Mr. Straub recently completed three years with the Connecticut Commission of Alcoholism, where he served as counselor and activities coordinator at the State Rehabilitation Center, New Haven.

A graduate of the Yale Summer School of Alcohol Studies, he has served as a consultant to the Onondaga Council on Alcoholism, Syracuse, New York. During his 15 years of experience in the field, Mr. Straub has lectured on alcohol to various medical, nursing, and civic groups.

In 1955, the Syracuse Council made an award to Mr. Straub in recognition of his outstanding contributions to their work. Similarly, the Pennsylvania Department of Health, in 1956, extended him recognition for his voluntary service in assisting with the alcoholism program in state institutions.

Mr. Straub's appointment is in accord with Melwood Farm's policy of providing experienced and capable staff members to carry out its program of understanding guidance on the problem of alcoholism.

Maryland
SOCIETY OF PATHOLOGISTS INC.
EDWARD C. MCGARRY, M.D., *President* MANNING W. ALDEN, M.D., *Secretary*
Annapolis, Md.

PAPER ELECTROPHORESIS

THE CLASSIC INVESTIGATIONS on electrophoresis, performed by Tiselius in 1937, showed that heterogeneous mixtures of charged molecules could be separated by differences in their mobilities in an electrical field. Separation of protein mixtures by this method has subsequently led to important findings of direct clinical application. This paper will undertake a description of the electrophoretic technique and a discussion of its clinical application and significance.

The classical method of Tiselius, which involves separation of proteins in free solution, is too cumbersome for the routine clinical laboratory; however, a simple and convenient method of electrophoresis, using filter paper as the supporting medium, has been developed. The sample is small; the filter paper is easily stained with various dyes to demonstrate protein patterns, and the experience needed is modest.

The principle of electrophoresis involves separation of proteins by means of an electrical current. The rate of mobility of each molecule will depend upon its size, shape, and total electrical charge, which can be varied by changes in the pH of the surrounding medium.

PART I

Serum Protein Electrophoresis

PAPER ELECTROPHORESIS at pH 8.6 separates normal serum proteins into five major components, of which albumin comprises 55 per cent. Globulins, which make up the remaining 45 per cent of the total proteins, are separated into four fractions: alpha$_1$ (α_1), alpha$_2$ (α_2), beta (β), and gamma (γ).

The four globulin fractions include antibodies, lipoproteins, mucoproteins, a variety of enzymes, elements of coagulation, and hormones. Most antibodies are contained in the gamma fraction; whereas the other specific globulins may occur in one or several of the remaining electrophoretic globulin fractions. Certain generalizations can be made, however, to permit interpretation of the findings in specific disease states. Consideration will be limited to the antibodies, lipo-

This is the first of two articles on Paper Electrophoresis. It is being printed on both sides of the page, so that you can remove this single sheet and place it in your file.

proteins, and mucoproteins, these being the substances responsible for most alterations in the electrophoretic pattern. Hormones, enzymes, and elements of coagulation are present in significantly lesser concentrations; and their demonstration by paper electrophoresis awaits further technical development.

Mild albumin deficiences are common in many diseases. Moderate deficiences occur with proteinuria, acute hepatic disease, ulcerative colitis, and malnutrition; while severe depletion is mainly attributable to massive proteinuria, chronic hepatic disease, and the rare exudative enteropathies.

A group of asymptomatic patients has been described recently with congenital absence of albumin, a condition referred to as congenital an-albuminemia. Paper electrophoresis of their serum proteins demonstrates, in addition to the absence of albumin, a marked increase in globulin fractions, which probably compensate for the loss of the oncotic effect of albumin.

The α_1 fraction, usually small, is composed primarily of the mucoproteins, which are a major constituent of the ground substance in connective tissue. Alterations in this fraction are not well understood, but elevations have been observed in acute infections and acute and chronic renal disease. Low values have not been correlated with clinical entities.

Alpha$_2$ (α_2) and β fractions are composed largely of lipoproteins, and disorders associated with abnormal lipid metabolism commonly affect these fractions. The greatest α_2 and β elevations are seen in the nephrotic syndrome, where the degree of elevation correlates well with the severity of the disease. The electrophoretic pattern of nephrosis is quite characteristic; there is 1) elevation of the α_2 and β fractions; 2) severe depletion of albumin; 3) moderate to marked depression of the γ fraction.

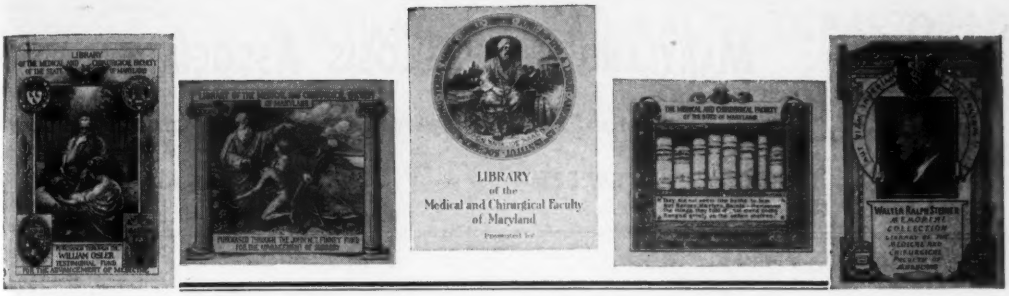
In hypothyroidism, moderate elevation of β globulin occasionally occurs. This finding is compatible with the abnormal lipid metabolism known to occur in these patients, as evidenced by hyperlipemia and hypercholesterolemia, the etiology of which is clearly secondary to hormone deficiency.

Proper liver function is essential for the normal regulation of lipid and protein metabolism. Impaired function results in alterations of lipid metabolism, reflected by elevations in α_2 and β globulin. Liver disease may also result in multiple defects in protein metabolism, the most consistent of which is hypoalbuminemia. In addition, the γ globulins consistently reflect the stage of liver disease, being normal or slightly elevated in the acute phase and markedly elevated in later stages of liver disease.

The gamma globulin fraction contains most of the circulating antibodies. Absence of this fraction is diagnostic of agammaglobulinemia.

Increase in the γ fraction is seen in collagen diseases, ulcerative colitis, chronic hepatic disease, and the chronic inflammatory diseases, including lymphogranuloma venereum, tuberculosis, sarcoidosis, leprosy, syphilis, and chronic osteomyelitis.

Abnormal proteins usually appear as relatively narrow bands in the α_2 to γ range and are usually associated with multiple myeloma, macroglobulinemia of Waldenstrom, or cryoglobulinemia. Specific identification of these proteins requires other physical-chemical or immuno-chemical methods. Macroglobulins are identified by ultracentrifugation or immunological technique. Cryoglobulins are defined by their precipitation in the cold; and the abnormal protein of multiple myeloma can be differentiated by the clinical course of the disease and, in most cases, by demonstration of Bence-Jones protein in the urine, either chemically or electrophoretically, as a narrow band migrating in the α_2 and γ region.



Library

Louise D. C. King, *Librarian*

"Books shall be thy companions; bookcases and shelves, thy pleasure-nooks and gardens." *Ibn Tibbon*

WOMEN PHYSICIANS

"The longer you can look back, the further you can look forward."

—Sir Winston Churchill, before the Royal College of Physicians, March, 1944

RECENTLY, WE READ an article on the changes wrought by the acceptance of women as employees of banks. We *have* come a long way from green eyeshades and paper cuffs. Such progress has also taken place in other business and professional fields where women have participated, and medicine is no exception.

The medical education of women on an equal footing with men has been a long, hard-fought battle in which the United States has pioneered. From Elizabeth Blackwell, the first academically trained woman physician in the United States, and trouser-clad Dr. Mary Walker, who held a commission as assistant surgeon during the Civil War, up to the present, the medical profession has become increasingly infiltrated with women. Today there is no gathering of the medical clan without representation from the weaker sex.

While we cannot attribute all improvements during the last 100 years to women, unquestionably, their influence, both tangible and intangible, has been felt. The practice of medicine gained another point of view in its ranks, perhaps more circumscribed initially, but certainly stimulating; and competition was augmented. These two facts alone, aside from the broader social implications, could well have implemented the tremendous strides made in the profession during the last century.

Because of public and professional prejudice and their native capacity for detail, many women have made their mark as investigators; Mary Putnam (Jacobi) in 1870, Florence Sabin, Alice Hamilton, Gladys Dick, and Maude Abbott, to mention a few. For these women to succeed in the face of opposition took courage, persistence, and a faith in themselves and their sex which is unnecessary today. It is well that we, who reap the benefits of pioneers, pause in our mad rush to read of their struggles, failures, and attainments and to give honor to those who have made our present life and work possible.

Suggested Reading:

Alsop, G. F.: History of the Woman's Medical College, Philadelphia. (1950)

Bluemel, Elinor: Florence Sabin. (1950)

Lovejoy, E. C. P.: Women Doctors of the World. (1957)

Mead, K. C.: History of Women in Medicine. (1938)

Any good general history of medicine.

MARYLAND TUBERCULOSIS ASSOCIATION
Christmas Seal Agency for State of Maryland
900 ST. PAUL STREET • BALTIMORE 2, MARYLAND

The Big Push Ahead for Tuberculosis Control
Arden House Conference

Kirby S. Howlett, Jr., M.D.*

IT HAS BEEN SAID that experience is the best teacher. What has experience in the treatment of tuberculosis taught us? It has taught me that best therapeutic results are achieved in places where the best therapeutic talent is concentrated. In the early years of this century these places were the major tuberculosis resorts; e.g., Saranac Lake, Colorado Springs, and the Arizona desert. In more recent years, including the entire era of effective antituberculosis chemotherapy, talent has been concentrated and best results achieved in tuberculosis hospitals and in the tuberculosis divisions of general hospitals.

Yet, as judged by the publicity which it has elicited, the whole Arden House approach is exemplified by the discussion of the conference in the February 1960 issue of the journal *Public Health Reports*. Here it is emphasized that the burden of administering the therapy by which tuberculosis is to be eradicated is likely to be carried increasingly by general practitioners. In fact, the entire article mentions the tuberculosis hospital as even a participant in the proposed campaign exactly one time. After discussing for three paragraphs treatment of patients by private practitioners, this statement occurs, quoted *in toto:* "Those who do not improve under the prescribed regimen need a change of drugs or *may* [italics mine] need hospitalization." I am not surprised that the author of this unsigned article elected to remain anonymous.

Since I was not present at the conference, I do not know to what extent subsequent publicity may have distorted the views of the conferees themselves. As judged by what I have read, however, Arden House publicity not only accepts but actually encourages the idea that home treatment by general practitioners is, by and large, the principal solution to the tuberculosis problem. Obviously, home treatment, in appropriate cases and at appropriate times, is an important weapon in our total armament. But to promote it among patients, among the families of patients, and among physicians without the discrimination which only long experience with tuberculosis can bring is, in my view, a fallacy which may actually impede the achievement of our objective—the eradicaton of tuberculosis.

The idea that effective treatment of tuberculosis is also an effective public health measure is scarcely new. To be most effective, from the point of view of both the individual patient and the public health, the best treatment possible needs to be applied promptly, efficiently, and persistently in every patient with active disease—and possibly

*Superintendent and medical director, Laurel Heights Hospital, Shelton, Connecticut. Address given at the Northeastern Tuberculosis Conference in Baltimore in October, 1960. Information on the Arden House Conference was presented in the July 1960 and October 1960 issues of this Journal.

BALTIMORE CITY HEALTH DEPARTMENT

HUNTINGTON WILLIAMS, M.D.
COMMISSIONER

P. O. Box 1877 Baltimore 3, Md. Plaza 2-2000: Extension 307

Learn To Do Your Part In The Prevention Of Disease

even in some without current stigmata of activity. To achieve such a purpose, it is obviously desirable to stimulate the deep interest of as many physicians as possible and to promote medical education in our field all along the line. But let us not kid ourselves; this is a long term project. Such wide interest cannot be evoked, and such education and understanding cannot be achieved overnight by the mechanism of high pressure publicity. Meanwhile, it behooves us to lean heavily upon those trained and experienced physicians, in and out of hospitals, who already have the interest and talent which effective treatment of tuberculosis requires.

It is fine to have a conference like Arden House occasionally, to give the job we are trying to do a stimulus and a lift. But it is equally important that we keep the lift within limits which will not take our feet completely off the ground.

The Maryland Trudeau Society will hold a scientific session on pulmonary disease at the Tidewater Inn, Easton, Maryland on Saturday, February 11, 1961. Additional information may be obtained from the Maryland Tuberculosis Association, 900 St. Paul Street, Baltimore 2, Maryland (Telephone: MUlberry 5-3881).

Infectious Syphilis Cases Continue

WHILE THE NUMBER of cases reported of infectious syphilis has dropped dramatically since the end of World War II, the disease is still far from being controlled. Evidence of this is the recent experience in Baltimore city. During the third quarter of 1960, July through September, 92 cases of infectious syphilis were reported. During the same period in 1959, only 40 cases were reported. This upsurge of cases continued into October.

As has been stated many times in the past, permanent control of the venereal diseases is not possible until sexual promiscuity is curbed. Nationally as well as locally, a definite relationship has been noted between the increasing number of illegitimate births and venereal diseases. Until these problems come under better control, it is necessary that a vigorous anti-venereal disease program be maintained.

The Baltimore City Health Department urges physicians not to overlook the duty of reporting venereal disease cases. Robert E. Farber, M.D., is director of the Bureau of Communicable Diseases. His telephone number is Plaza 2-2000, extension 787.

Huntington Williams, M.D.

Commissioner of Health

A MENTAL HOSPITAL INSTITUTE FOR COMMUNITY PHYSICIANS

under auspices of

■ SPRING GROVE STATE HOSPITAL ■

■ THE MARYLAND ACADEMY OF GENERAL PRACTICE ■

■ THE PSYCHIATRIC INSTITUTE OF UNIVERSITY HOSPITAL ■

■ THE BALTIMORE PSYCHOANALYTIC INSTITUTE ■

■

Every Wednesday afternoon, March 1 to April 5, 1961, inclusive, 2 to 5 p.m.
at
The Hamilton Building, Spring Grove State Hospital, Catonsville, 28, Md.

■

Approved by AAGP for Category 1 Credits

■

PROGRAM

March 1, 1961 Welcome—Bruno Radauskas, M.D., Superintendent, Spring Grove State Hospital

Introductory Remarks—Isadore Tuerk, M.D., Commissioner, Department of Mental Hygiene of Maryland

Problems of Community Physicians in Relation to the State Psychiatric Hospital—Eugene I. Baumgartner, M.D., Secretary, Section on General Practice, American Medical Association

Before the Patient Comes to the Hospital—Riva Novey, M.D., Instructor in Psychiatry, Johns Hopkins University Medical School; and Mrs. Hubertine Marshall, Case Work Supervisor, Out-patient Services, Spring Grove State Hospital

March 8, 1961 **Current Concepts in Psychopathology (Diagnostic Categories)**—Allan R. McClary, M.D., Instructor in Medicine and Assistant Professor of Psychiatry, Johns Hopkins University; Chairman of Post-graduate Education Committee, Baltimore Psychoanalytic Institute

Chronic Brain Syndrome—E. T. Lisansky, M.D., Associate Professor of Medicine and Assistant Professor of Psychiatry, University of Maryland Medical School

March 15, 1961 **The Alcoholic Patient**—Isadore Tuerk, M.D.

The Ambulatory Psychotic Patient—Jerome Hartz, M.D., Assistant Professor of Psychiatry, Johns Hopkins University Medical School, Associate Professor of Psychiatry, University of Maryland

March 22, 1961 **Current Psychiatric Therapies I**

In Private Practice—Allan McClary, M.D.

In Psychiatric Hospitals—Bruno Radauskas, M.D.

March 29, 1961 **Current Psychiatric Therapies II**

Milieu Therapy—The Therapeutic Community—Aristides Simopoulos, M.D., Acting Clinical Director, Spring Grove State Hospital

Psychopharmacology and Somatic Therapies—Albert Kurland, M.D., Director of Research, Department of Mental Hygiene of Maryland, Director of Medical Research, Spring Grove State Hospital

Tour of hospital areas

April 5, 1961 **The Post-Hospitalized Patient**—Gertrude J. Fleischmann, M.D., Director of Out-patient Services, Spring Grove State Hospital; Mrs. Hubertine Marshall

Evaluation of Institute

Demonstration interviews, discussions, question and answer periods, etc., will be included in the program.

Application for enrollment can be obtained from Bruno Radauskas, M.D., Spring Grove State Hospital, Catonsville, 28, Maryland. Registration fee: $25.00

The Heart Page

Luis F. Gonzalez, M.D.--*Editor*

A SERVICE OF

THE HEART ASSOCIATION OF MARYLAND

Pathological Lesions in the Heart Secondary to Myocardial Infarction

Luis F. Gonzalez, M.D.

THE OCCLUSION of a coronary artery with infarction of the myocardium usually results in a localized area of scar tissue with satisfactory functional capacity. More serious anatomical changes may occur, however. Two of these, ventricular aneurysms and myocardial rupture, will be discussed.

Ventricular aneurysms have been found in 10 to 38 per cent of patients who have had severe acute infarctions. Shortly after the occurrence of an acute myocardial infarction, it is possible to demonstrate gross paradoxical pulsations of the damaged area. The formation of aneurysms occurs frequently as a late complication of extensive infarcts. The prognosis depends on the extent of the myocardial lesion, the degree of coronary artery disease, and the condition of the rest of the myocardium. Pulmonary hypertension and decrease in cardiac output may occur in many of these patients. Lesions of sufficient size to cause impairment of cardiac function should be repaired, as emphasized by Cooley (Ann. Surg. **150**:595-612, 1959).

Rupture of the interventricular septum may occur as a complication of myocardial infarction. It can be detected clinically by the sudden appearance of a loud systolic murmur with thrill, not previously present in a patient with myocardial infarction. Intractable congestive heart failure may be prominent and difficult to treat medically. Reconstructive surgery in a few selected patients has been reported recently.

An almost universally fatal complication of myocardial infarction is rupture of the ventricular wall. It has been found in 7 to 15 per cent of series of cases of myocardial infarction which have come to autopsy. Correlation of the clinical and autopsy material shows that rupture tends to be more prevalent four to 11 days after an acute myocardial infarction, usually in an hypertensive patient with no previous history of myocardial infarction or congestive heart failure. The hypertensive state tends to persist through the hospitalization period, and unusual effort has often occurred within 24 hours prior to rupture. At autopsy, the patient with cardiac rupture usually has little evidence of additional serious heart disease, other than the stigmata of hypertension. This should stress the importance of adequate rest in patients with acute myocardial infarction. Rupture seems to be likely in the patient with apparent mild, uncomplicated infarction, in whom limitation of activities does not appear mandatory and whose blood pressure does not drop appreciably following infarction. Whether or not anticoagulants contribute toward any increased incidence of myocardial rupture following myocardial infarction is still debatable.

Woman's Auxiliary
Medical and Chirurgical Faculty
MRS. E. RODERICK SHIPLEY *Auxiliary Editor*

JANUARY, 1961

HIGHLIGHTS OF THE CHICAGO CONFERENCE

Mrs. Norman Oliver

THE SEVENTEENTH ANNUAL conference for state presidents, presidents - elect, national officers, and chairmen was a most rewarding experience. The meeting started with the get-acquainted membership dinner on Sunday, October 2, at which the National Auxiliary was host. Membership activities and projects were informally discussed. The general atmosphere of friendliness was so prevalent that those of us who were there for the first time soon forgot our feelings of apprehension.

Some of the outstanding issues that were presented were:

1. The importance of being a well-informed member of our Auxiliary and the need to convince the disinterested doctor's wife to join. Each member has a wonderful opportunity to inform others of our interest in promoting better understanding for the advancement of medicine and public health.
2. The importance of supporting the American Medical Education Foundation. The national chairman would like for every county auxiliary have one fund raising affair for this purpose.
3. The need to encourage and guide intelligent young students into a health career, where they can carry on our great traditions and contribute to the needs of the future.
4. The great need to be informed about civil defense and to have our homes prepared.
5. The importance of subscribing to and reading the *Bulletin*. Auxiliary members will find the answers not only to their questions but also to the questions others in the community might ask.
6. The presentation of *Today's Health* subscriptions to organizations and individuals. This is an excellent way of bringing the views of medicine to the general public.

The early bird discussion of parliamentary procedures, under the able direction of Mrs. William Mackersie and Mrs. Mason G. Lawson, was invaluable. This was a demonstration of parliamentary procedures based on questions submitted for discussion. The 8:00 a.m. meeting time kept few away. I hope this will be on the agenda again next year.

Mrs. Louise Stone, our State president, and Mrs. Albert E. Goldstein also attended the meeting.

THE MONTGOMERY COUNTY Auxiliary invites you to their lavish and exciting fashion show on January 31. It will be held in the beautiful Shoreham Hotel Blue Room and will feature fashions by Julius Garfinckel and Company.

In addition to the luncheon and fashion preview, guests will have the opportunity to meet Mrs. R. Messenger, well known fashion commentator, and Frank Harden, of WMAL's "Harden and Weaver" show, who will be our master of ceremonies.

Tables will be set up for ten. Tickets are $6.00 each and are available from Mrs. Peter A. Santucci, 5815 Ogden Court, Bethesda 16, Maryland, phone Oliver 6-3186. Proceeds will go to our nurses scholarship fund.

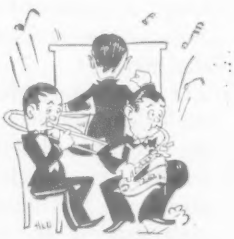
Plan to drop everything on the last cold day of January and send us your check for $6.00, or, better still, make up a table of your friends.

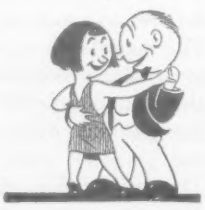
COME

to the

Mardi Gras Ball

on Saturday, February 4, 1961 at the Blue Crest North. Champagne Hour starts at Seven, Dinner at Eight. Then Dance till One to the Music of Rivers Chambers. Tickets are Ten Dollars a Person. Send your Check to Mrs. Sullins G. Sullivan, 419 Oak Lane, Towson 4, Maryland. Or make your Reservation by Phoning Mrs. Raymond L. Markley at VAlley 5-9709. This is the annual Dinner Dance sponsored by the Woman's Auxiliary to the Baltimore City Medical Society to aid Medical Education. Have a Ball. Come to the Mardi Gras.





CIVILIAN DEFENSE SEMINAR

"Survival of the knowingest—not the fittest"

An all day seminar on civilian defense techniques was held at the Pikesville National Guard Armory on October 19. It was sponsored by the Woman's Auxiliary to the Medical and Chirurgical Faculty and the Baltimore County Medical Society. Mrs. Charles H. Williams, Civil Defense chairman for the State Auxiliary, was in charge of the program.

Charles H. Williams, M.D., and Kathleen Jones, R.N., talk about mouth-to-mouth resuscitation.

I. Ridgeway Trimble, M.D., brought greetings from the Faculty. He warned that the matter of civilian defense should not be treated with pure apathy; however, some heartening advances have been made recently. The budget allowed nationally for civilian defense is about sixteen million dollars, a sum less than that allotted to the District of Columbia zoo. Dr. Trimble quoted Secretary of State Herter as saying, "Those able to put the populace in a safe place in a short period of time in the event of atomic warfare are in the better bargaining place." Dr. Trimble added that civilian defense needs the full time efforts of a well trained organization; we can't wait for the government to start on this course, but must push for it from the bottom up.

Dr. and Mrs. Charles H. Williams help a reporter get the facts.

Navy Nurse Kathleen Jones demonstrates how the mannequin bleeds.

Miss Blair Stewart, R.N., from the Norfolk civilian defense office, gave a tremendously interesting account of a program initiated in the Norfolk area with the help of the Naval Training Station. She related in detail the operation of a program of compulsory mass training in high schools. The idea was broached by health and physical education instructors at a nursing meeting.

The Board of Education gave its approval, and the project was launched. Completion of a basic first aid course was the primary prerequisite for enrolling in the casualty course. A mannequin was bought for demonstrating to students the recommended procedures. The mannequin, designed by a navy dental technician, bleeds and simulates 200 possible injuries to the body. It is completely articulated with many movable parts to make teaching more simple. Weighing half a ton, the mannequin is carried about on a special stretcher and transported by truck to each of the schools where the course is in progress.

Miss Stewart was accepted for training at the Norfolk Naval base so that she could oversee the course for the schools. The course covers injuries from atomic blasts, burns, radiation, and psychological reaction. Miss Stewart divulged that by the end of 1960, Russia will have trained 5,000,000 school children in casualty care. In the first year that the course was offered in the Norfolk area, 49,000 high school pupils finished the course. A proposal has been made to offer it in junior high schools as well. As a parting thought, Miss Stewart cautioned that poor casualty care can be worse than none at all.

Miss Blair Stewart shows how mannequin aids in training programs.

Miss Alice Smith, R.N., chief of nursing, Washington Missionary College, talked about communist influence in this country. Brain washing is an applied science as taught to the communists. Pavlov's experiments with the rats in the maze were the basis for the theories they developed, perfected, and taught to those members of the party who are entrusted with spreading the communist ideology throughout the world. Miss Smith disclosed the fact that there are 15,000 hard core communists in this country, whose mission is to create confusion, to indoctrinate the unwary, to promote an attitude of mass conformity, and to discourage individual effort. Their line of persuasion is directed toward trying to change Americans from individuals to conformists. They preach "peace at any price," "try to get along with all," and "what's in it for me?" attitudes instead of "what can I give for my country?" Miss Smith summed up her message with the emphatic statement that the children in our schools need to be taught about freedom, our country, and its heritage.

Charles H. Williams, M.D., in explaining mouth to mouth resuscitation, illustrated how our lungs work by affixing balloons to the ear tubes of his stethoscope and blowing into them as one would breathe. He then proceeded to demonstrate mouth-to-mouth resuscitation on an inflatable dummy, borrowed for the occasion from the Edgewood Arsenal. He showed first the technique using his mouth alone and then the use of the resuscitube. While Dr. Williams worked, a nurse from the Navy Nurse Corps explained preparation of the patient and listed the precautions concerning tight clothing, dental appliances, and vomiting.

A film on mouth-to-mouth resuscitation, sponsored by Walter Reed Army Hospital and filmed at Baltimore City Hospitals, graphically illustrated the procedures and precautions that had been explained by Dr. Williams and the nurse.

Mr. J. Henry Schilpp, public health engineer, Bureau of Environmental Hygiene of the State Department of Health, spoke on securing a safe water supply. We can ill afford to be unprepared, he asserted. Environmental health is concerned with preventive medicine, and the radiological service was recently added to this branch of the public health program. In the prevention of death, water supply is the most critical element. We cannot count on being forewarned, so it is imperative that everyone be sure of a 14 day water supply by storing seven gallons of water or other fluids for each member of the family. Containers should be rinsed out and refilled every three months. For emergency use, wipe them clean before opening them. Turn off the water to your house, for the water already in the pipes will be clean unless it is contaminated by incoming dirty water. Liquids in the refrigerator, melted ice cubes, the bathroom flush tank, and the water heater are sources of fluids in an emergency. Diatomaceous earth filters and water softeners remove radioactivity.

Mrs. Carl Wheelock, steering committee, Office of Civilian Defense, Region Two, discussed home preparedness. She brought with her a kit that may be ordered from the local Civil Defense Office, which contains instructions for preparing one's home for a disaster shelter and a list of necessary items for stocking the shelter for a two week period. Families which comply with the regulations as outlined in the kit will receive a sticker to be displayed in a window of their home. A knowledge of which homes and other places that are prepared will be invaluable in case of an actual disaster.

Mr. Cosmo Liberti, Chemical and Biological Warfare, Office of Civilian Defense, Region Two, elaborated on radioactive fallout. Fallout is minute radioactive particles that result when an atomic bomb explodes. Fallout comes only from the atomic bomb, which is now used to detonate the hydrogen bomb; no substitute has yet been found. We can protect ourselves from fallout, but the duration of radioactivity is long. Areas up to 7.1 miles from the blast are destroyed. For protection, have an adequate fallout shelter—not a bomb shelter—well stocked for a two week survival period. Our only salvation from the bomb itself is the good fortune to be someplace else. Mr. Liberti also reminded us that we can distill water for emergency use. Distilled water is pure.

A low-priced luncheon was served at a local church. In the afternoon, a tour of the shelter in civilian defense headquarters at Pikesville was conducted.

This was an informative program and was extremely well attended. It is serving as a model for similar seminars in other regions.

V.G.S.